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DIAGNOSIS AND CLINICAL FEATURES OF POLIOMYELITIS.[1]

By STANLEY WILLIAMS, M.D.,
Melbourne.

WHEN a similar symposium to the present one was presented in Melbourne during the epidemic of 1937-1938, the diagnosis of poliomyelitis was considered reasonably straightforward, and many cases of non-paralytic poliomyelitis were diagnosed. Uncertainty in the presence of physical signs alone was the feeling in the epidemic in 1949, and now we are even more uncertain. It has been shown that poliomyelitis is only one cause of meningo-encephalitis, and other causes are perhaps more common than has hitherto been realized. Meningo-encephalitis is the term applied to that group of intracranial infections which does not include pyogenic meningitis. The Coxsackie virus can produce meningo-encephalitis, as also can the virus of mumps, measles and chickenpox. There is a meningo-encephalitis associated with glandular fever, and I learn from Dr. A. Ferris of his findings in leptospirosis. For practical purposes, poliomyelitis is the only disease of the meningo-encephalitis group that produces a lower motor neuron type of paralysis. As it is the most important cause of meningo-encephalitis, and as the subject of poliomyelitis is the feature for this symposium, it will be assumed that when meningo-encephalitis is diagnosed, poliomyelitis is the most likely cause—provided that measles, mumps and chickenpox have been excluded.

Age.

The youngest patient with paralysis due to poliomyelitis in my series was aged seven months, and the oldest was aged sixty-four years. At Fairfield Hospital in 1949 there were four infants aged five months.

Clinical Features of Poliomyelitis.

To discuss the clinical features and diagnosis of poliomyelitis, let us consider a typical case which results in paralysis. A child or adult will complain of malaise and headache on the first day, and will have a fever, and possibly will complain of a sore throat or abdominal pain. Pain in the back of the neck follows, and soon also pain in the region of the lumbar part of the spine. There will be an apprehensive appearance, tachycardia, neck stiffness and spine stiffness. Tremor of a limb may follow in a few hours, perhaps pain in the shoulders or thighs, and next day paralysis is noted. The time from the onset to the appearances of paralysis is one to seven days. Increasing paralysis may continue to be detected daily over the next two or three days, and the full extent of muscle involvement may not be known for two to three weeks.

A more detailed description of the above-mentioned symptoms and signs will help in differentiating poliomyelitis. The headache is frontal in position. The pain in the neck and spine is in the mid-line, and at the onset of the disease

[1] Part of a symposium on poliomyelitis arranged by the Melbourne Permanent Post-Graduate Committee in conjunction with the Consultative Council for Poliomyelitis, held on March 29, 1952, at Fairfield Hospital.

there is seldom pain away from the mid-line. Tenderness in the muscles is not a feature at this stage. The neck stiffness and spine stiffness, once they appear, persist for many hours, and even in those cases presumed to be poliomyelitis in which paralysis does not develop, the neck stiffness may persist for two or three days. The neck stiffness persists for periods varying from twelve hours to four days in the moderately sick poliomyelitis patient who becomes paralysed. Examination for these signs must be cautiously undertaken in the case of young children or infants, who readily become suspicious when their head is held and when they are inclined voluntarily to resist forward flexion. The placing of the hands under the pillow, or on each side of it, as shown in Figures I and II, may reduce the child's voluntary resistance. The spine test can be carried out by asking the patient to sit up. His hands are placed to the side to help support the painful spine, a tripod being thus formed. However, a sick child or adult with headache from any cause dislikes this manœuvre, and the test for spine stiffness can be made with the patient lying on his side, having just rolled over on the bed with the head still supported on the hand or pillow. The patient is asked to approximate

FIGURE I.

Testing a child for neck stiffness. Many children voluntarily resist when a hand is placed under their heads. This voluntary rigidity is reduced if the examiner's hand is placed under the pillow.

the head to the knees; assistance can be given towards this. If there is spine stiffness he is unable to bring the head very close to his knees. The slightest spinal flexion may be painful.

Onset of Paralysis.

The onset of paralysis is the only confirmatory sign of the diagnosis of poliomyelitis.

Inability to move the limb or muscle suspected of being paralysed is the usual method of diagnosis. A weakness can often be detected by testing active muscle movement, perhaps against the resistance of the examiner's hand—for example, in flexion and extension of the upper and lower limbs. When possible, elicit the deep reflexes as a guide. This can be done in the examination of the youngest infants. The knee, ankle and biceps jerks are very helpful in reassuring the examiner that the muscles in those areas are not affected. The triceps jerk is less reliable if it is absent or weak.

Lumbar Puncture.

Examination of the cerebro-spinal fluid is very helpful in confirming or helping in the diagnosis. The presence of white blood corpuscles varying in number from 20 to 500 per cubic millimetre, and mostly lymphocytes, is usual. In the early stages there may be a preponderance of polymorphonuclear cells. In most instances the protein content is increased. The sugar and chloride contents are normal.

Atypical Onset.

An atypical onset may herald the illness of poliomyelitis, in both epidemic and non-epidemic times, in one of several ways.

1. During the epidemic of 1937 occasionally the often described biphasic or dromedary type of temperature chart was seen, with two periods of fever and three to four days of normal temperature in between. Paralysis followed the second wave of pyrexia.

2. A mild type of illness with fever, but without headache, neck stiffness or spine stiffness, yet followed by paralysis, has often occurred. However, it could be suspected in the early stage only if a routine lumbar puncture was carried out in all cases in which pyrexia of unknown origin was present.

3. A febrile illness accompanied by muscular pain of the fibrositic type, with the pain and tenderness at a site away from the mid-line, would not ordinarily cause one to suspect poliomyelitis. This type of onset has been seen with the development of paralysis, and no spine or neck stiffness discernible. When it has occurred, the patients have usually been aged over forty years.

FIGURE II.

Testing a child for neck stiffness. A modification of the method described in Figure I is for the examiner to hold the ends of the pillow, and whilst engaging the child's attention, to attempt gentle flexion of the head by lifting the pillow.

4. The bulbar region may be the only area affected, and although the premonitory signs and symptoms before the onset of paralysis are the same as those described above for the typical case, the disease may not be suspected until respiratory and swallowing difficulty point to poliomyelitis as the cause of the illness. Difficulty in swallowing and in breathing, gasping respiration, gross accumulation of saliva and regurgitation of fluid through the nose, are signs of severe disease. It has been the experience of many that these signs may be present for many hours or even days before it is realized that the illness is poliomyelitis and not a respiratory infection. Paralysis of the muscles of respiration is more readily detected than pure bulbar paralysis, and paralysis of the intercostal muscles is usually accompanied by involvement of some of the muscles of the upper limbs.

Differential Diagnosis.

The differential diagnosis has included dozens of instances of infections or other diseases which have produced meningismus, meningitis, meningo-encephalitis, paralysis or pseudoparalysis.

In the history and general examination inquiries will have been made to attempt to exclude measles, mumps and chickenpox or *herpes zoster*. The diagnosis of tuberculous meningitis is considered in the light of a history of contact, a history of the disease itself and the findings on lumbar puncture.

"Pseudoparalysis", which is actually a disinclination to move a limb or muscle because of pain, may be due to trauma such as a fracture of a bone, or a bruise. The same applies to rheumatic fever and to arthritis from other causes. An abscess in the joint, such as "Smith's" arthritis in very young infants, may at first appear to be paralysis. Scurvy is a not uncommon cause of pseudoparalysis, but congenital syphilis is extremely rare.

Of the diseases that result in lower motor neuron paralysis, in a recent series of 125 patients examined in consultation, when poliomyelitis was suspected by the local doctor, there have been two cases of peripheral neuritis. The differentiating feature in these patients is the absence of cells in the cerebro-spinal fluid and the increased protein content. Disseminated sclerosis was the diagnosis in one case, and two patients had spinal tumours. Diphtheritic paralysis was the cause in two cases, and another patient had paralysis of the deltoid, associated with *herpes zoster*.

Perhaps the most difficult group of patients from the point of view of differential diagnosis are those with pains in the muscles and fever, and headache. Although some of these may have meningo-encephalitis, it is unlikely that they will go on to develop serious poliomyelitis if they do not exhibit persistent neck stiffness. Especially in non-epidemic times, as at present, they are not regarded as suspect poliomyelitis patients.

Summary.

The early signs and symptoms of meningo-encephalitis are similar, whether the infection is due to poliomyelitis, mumps, measles, chickenpox and possibly other virus infections, such as those produced by the Coxsackie virus.

Poliomyelitis is the only disease of that group that may produce a lower motor neuron type of paralysis.

The diagnosis of meningo-encephalitis is made on the history of headache and malaise, and on the signs of persistent neck and spine stiffness.

A lumbar puncture may reveal changes in the cerebro-spinal fluid typical of poliomyelitis. If the clinical features are not typical of poliomyelitis, diagnosis is possible only on the development of paralysis, or by lumbar puncture.

There are many patients who do not develop paralysis in whom the diagnosis of poliomyelitis is suspected, but in the absence of complete laboratory investigation for poliomyelitis, which is not yet available in Australia, an accurate diagnosis is impossible. However, the condition of an ever-increasing number of patients, who at one time would have been regarded as having non-paralytic poliomyelitis, is being more correctly diagnosed by improved laboratory methods.

THE MANAGEMENT OF THE ACUTE PHASE OF POLIOMYELITIS.[1]

By H. McLORINAN,
Medical Superintendent, Fairfield Hospital, Melbourne.

IN the management of the acute phase of poliomyelitis we are influenced by two important observations: (i) that of Ritchie Russell (1949), who noted the dangerous effects of muscle fatigue; (ii) that of McCloskey (1951) and of Bradford Hill and Knowelden (1950) on the harmful effects of certain injections.

The implication of these observations is that these two "provoking factors" (they are certainly not the only ones) act as insults not only to the muscle itself, but to the corresponding nerve cells probably through the afferent and efferent impulses from the muscle. The practical application of these observations in the management of an early case of poliomyelitis is then as follows: (*a*) The necessity for complete physical rest once symptoms have developed. There is some evidence to show that the most dangerous period is in the second or meningeal stage of poliomyelitis. (*b*) The necessity for care in the examination of the patient. Muscle testing against resistance, standing out of bed for the diagnosis of a limp *et cetera* are contra-indicated. (*c*) The avoidance of multiple injections into muscles. (*d*) The judicious use of sedatives. Although complete physical and mental rest is the ideal to be aimed at, there is a risk that heavy sedation may so depress the respiratory mechanism that in the event of respiratory paralysis the result could be disastrous.

With these precautions borne in mind, the treatment otherwise follows fairly general lines. There is no specific drug therapy that has any influence on the course of the disease.

The bed should be firm and we usually use a fracture board. A sagging bed or mattress can cause much pain. Relief of pain, which may become very severe after paralysis has developed, may be accomplished by analgesics or the application of various types of heat. Immediate splinting is rarely necessary; but care should be taken to avoid the stretching of weakened muscles over a long period. The use of sand-bags, pillows, slings and plaster casts is necessary to avoid malformation and subsequent development of deformities. Usually within a fortnight of the onset, however, some form of rigid splinting should be applied. The much discussed stiffness and spasm of muscles can be diminished by early passive movements; but I am of the opinion that no great harm is done by delaying active movements for about a fortnight. The Hubbard bath is helpful both psychologically and physically, and bathing should be commenced as soon as the patient can be moved, without much pain, after the temperature has subsided. Retention of urine is often a troublesome complication. There is some evidence to show that "Prostigmin" is helpful in this condition.

Bulbar Paralysis.

We now come to the management of patients with bulbar paralysis. The patient with true bulbar paralysis has symptoms related to the ninth, tenth and eleventh cranial nerves, although sometimes the facial nerve is involved as well. Involvement of these cranial nerves is indicated by paralysis of the palate, the pharyngeal muscles and the laryngeal muscles, with resultant difficulty in swallowing, a weak ineffective cough and the pooling of secretion in the respiratory tract. Respiratory obstruction soon becomes evident. Occasionally also the respiratory and cardio-vascular centres in the medulla become affected. The treatment is directed towards the relief of the respiratory obstruction, and consists of the following measures: (i) suction of the pharynx, (ii) postural drainage, (iii) oxygen therapy, (iv) tracheotomy, (v) intragastric feeding.

If signs of medullary respiratory failure develop, or if the bulbar signs are associated with high spinal paralysis involving the intercostal muscles and the diaphragm, the patient has also to be placed in a respirator. When a patient has pharyngeal paralysis with pooling of mucus, it is advisable to perform a tracheotomy before using the respirator.

Respiratory paralysis due to involvement of the intercostal muscles and the diaphragm occurs in the spinal type of poliomyelitis, and is usually associated with extensive paralysis of limbs and trunk. This type of case nowadays mostly occurs in young adults, particularly in the twenty to twenty-four years age group, and it is responsible for this group having the highest mortality figures in Victoria. We have noted this type frequently in farm workers, the nature of whose occupation often demands keeping on working until symptoms are well advanced.

It is a difficult matter to decide when to put a patient in a respirator. It may be suggested that the respirator should be used early in order to rest the respiratory muscles. This sounds well in theory, but in practice the results are not satisfactory. For some reason the respirator often seems to make these muscles worse. Perhaps the respiratory muscles, which are used to working

[1] Part of a symposium on poliomyelitis arranged by the Melbourne Permanent Post-Graduate Committee and the Consultative Council on Poliomyelitis on March 29, 1952, at Fairfield Hospital.

twenty-four hours a day automatically, do not become fatigued like other muscles. Whatever the reason, it is our custom not to use the respirator until the patient is showing signs of exhaustion and is using the accessory muscles of respiration. This type of patient, when finally placed in a respirator, usually synchronizes well and often falls asleep.

The best results with respirators are obtained in children. Those aged under fourteen years usually survive, but strangely very few children require respirators these days. Adult females do not do well, but they do better than adult males. This suggests that the more rigid muscular chest wall of the adult makes it more difficult for the respirator to provide an adequate gaseous exchange.

To those of you who are not familiar with this type, I would like to describe the course of a typical case. A young healthy adult is admitted to hospital with a history, usually of the biphasic type of poliomyelitis. He frequently has no weakness, except perhaps slight weakness of the triceps of one arm. In a short time this extends to the other arm, an occurrence which always fills me with dread. A few hours later he complains of a heaviness across the chest and may describe it as being like an iron band. The cough becomes weak, the movements of the chest and diaphragm become visibly feebler, and the patient is unable to say more than two or three words with each breath. With consciousness quite unimpaired, many patients are obviously aware of what is happening, and their apprehension is one of the most unpleasant features of the disease. I myself loathe and detest these cases, and I long for the day when either they can be prevented or the patient can be cured. Ultimately the respirator is used and the patient for a while is comfortable, and for anything from six to twenty-four hours he appears quite well. For that time both the relatives and the patient are comparatively happy; but later the patient complains of increased secretions in his respiratory tract, and very soon, in spite of posturing and suction, he has obvious obstruction. He now becomes restless and delirious. The symptoms are again obviously anoxic, and tracheotomy is performed with the patient lying on the stretcher of the respirator and given oxygen under pressure. After considerable secretions have been aspirated from the larynx and bronchi, temporary relief is obtained. Soon, however, the secretions accumulate again, and even after bronchoscopic suction the patient's condition is as bad as ever and finally he dies. The post-mortem examination usually reveals extensive collapse in the lungs and frequently bronchopneumonia. It is frankly admitted that the results of respirator treatment in the male adult group are bad; but it would be wrong to condemn all respirator treatment on this account. We have had many brilliant and worthwhile "saves". Bower and his co-workers (1950) in Los Angeles claim to have obtained improved results by using a synchronized apparatus giving oxygen under positive pressure by the intratracheal route either by way of a face mask or through a tracheotomy tube. They maintain that these patients suffer not so much from anoxia as from poor gaseous exchange with resulting carbon dioxide retention and pulmonary acidosis. There is no doubt that we have still much to learn about respirators, particularly in the treatment of adults, and the services of the physiology school must be requisitioned.

Points in Nursing.

Finally, I should like to mention some special nursing points in the management of the acute stage. It is important to watch for the following: (i) gastric dilatation with vomiting of dark brown fluid material and rapid dehydration (this may occur in patients with respiratory weakness; treatment is by immediate suction of gastric contents); (ii) urinary retention or overflow incontinence; (iii) obstinate constipation with impacted fæces.

But, to sum up, the main points in the treatment in the acute phase are as follows: (*a*) physical and mental rest; (*b*) the maintenance of an airway and adequate gaseous exchange in the bulbar and respiratory types.

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THE PREVENTION OF CRIPPLING FOLLOWING POLIOMYELITIS.[1]

By JEAN MACNAMARA,
Melbourne.

THE invitation to take part in this post-graduate day gives me an opportunity to pay tribute to Fairfield Hospital; to acknowledge the contribution which the staff of this hospital has made in shepherding hundreds of Victorian patients, paralysed by poliomyelitis, along the first, most difficult, part of their road to complete recovery or to a useful life.

I have had many opportunities to learn of the humanity which is characteristic of Fairfield Hospital. Observations in other countries have shown me that any community plan for the after-care of paralysed people is handicapped from the start unless there is close liaison between the doctors responsible for the patients during the acute stage and the team caring for the same patients after their discharge until recovery or rehabilitation is complete.

As the honorary medical officer responsible for the Itinerant Physiotherapy Service of the Children's Hospital, which has cared for paralysed Melbourne children in their homes since 1931, I have reason for gratitude to Fairfield Hospital. Almost all the children have been measured for and fitted with, and have become accustomed to, their frames before their discharge to after-care. It has been easy to discuss any modification of policy which would make after-care problems simpler. The parents and the patients have been prepared at Fairfield Hospital for the programme ahead.

As a result of the kindliness shown them and the interest taken in each child—in his general welfare and in his paralysed muscles—parents have been receptive to the help which the Children's Hospital endeavours to commence on the day following the patient's discharge to after-care. They appreciate that the team is aiming high and is not content with a standard at which the child can get about in some fashion with an ugly limp; but, the disease poliomyelitis having done its worst, the future usefulness and happiness of the patient depend largely on the details of his care in the programme ahead.

The influence of Dr. Harry McLorinan and of Dr. F. Scholes over many years has resulted in an attitude of parents expecting, and patiently cooperating in, the care planned to achieve the greatest possible recovery.

It is good that the Melbourne Permanent Post-Graduate Committee has regarded poliomyelitis as a subject whose importance merits a whole-day session. In a country short of man-power, stressing the need for production, especially primary production, muscle power is valuable. Australia already carries a heavy burden in taxation to provide invalid pensions. Therefore, measures to salvage persons paralysed by poliomyelitis, to give muscles capable of recovery the optimum opportunity to recover, are matters of national importance.

[1] Part of a symposium on poliomyelitis arranged by the Melbourne Permanent Post-Graduate Committee in conjunction with the Consultative Council for Poliomyelitis, held on March 29, 1952, at Fairfield Hospital.

The change in the type of paralysis caused by poliomyelitis which has occurred in the past thirty years makes careful, continued after-care more imperative now than it would have been early in this century. When paralysis was severe, often total, in the muscles of one limb, the rest of the musculature escaping, the place of after-care was less important than it is at present, when paralysis is frequently diffuse, generalized and partial. A high proportion of the crippling which followed poliomyelitis thirty years ago could more fairly have been attributed to the disease poliomyelitis. Now the disease leaves many more potential cripples than actual cripples. It is the responsibility of the community and of doctors to prevent the manufacture of actual cripples from those whom the disease poliomyelitis has hurt, but not crippled.

Post-graduate instruction is more necessary for poliomyelitis than for many other subjects covered adequately in the medical curriculum. I have listened to Melbourne graduates, practising in other States, complain that in practice they have had to meet medical emergencies such as bulbar poliomyelitis, having had little opportunity to learn the handling of these patients; or, troubled at failure to recognize paresis of certain muscles, they have regretted that, as students, there had been little opportunity for them to learn how to detect relative loss of power of muscles and, by that recognition, to be able to save their patients the additional weakness which follows activity and fatigue, especially by weight-bearing on a weakened lower limb.

At present, one two-hour session covers the Melbourne student's opportunity to learn how to examine patients for muscle weakness, and something of the principles of care. At Aberdeen the student's interest in the meaning and economic significance of the action of muscles is stimulated in his study of anatomy. More interest in "living anatomy" early in the medical course would make for better appreciation of the handicaps which result from weakness, or from lowering of the fatigue point of certain muscles of individual muscle groups.

For example, one of my patients recovered quickly from the paralysis which had involved his right deltoid and several muscles of his right lower limb. He was allowed to stand three months from the onset; weight-bearing and walking were cautiously increased until it was obvious that neither the deltoid nor the muscles affected in the lower limb would be pressed past their fatigue point by return to work. I had considered that the power of his right hand had not been affected, and so had the patient. Soon after his return to work he complained of disability in a task his work demanded—that of using a long fine screw-driver for several hours each day. It was obvious, on examination, that the first dorsal interosseous muscle of his right hand had wasted since his work commenced. The index finger deviated to the ulnar side at the metacarpo-phalangeal joint when he was holding his screw-driver. Had my early examinations of the power of the individual muscles of his right hand been more thorough, and had inquiries into his tasks at work been more detailed, he could have been saved this set-back to rehabilitation which followed the fatigue of this small muscle.

Power quickly returned to the "sulky" first dorsal interosseous muscle when the hand was splinted in a position to favour the muscle, the splint being worn day and night for a month and then worn when he was not at work for several weeks, and afterwards for a long period for protection at night only.

The Aberdeen students who posed for Professor R. D. Lockhart's "Atlas of Living Anatomy" (1948) would be able to carry out detailed muscle examinations more easily than would those of medical schools with a cadaveric bias. It is also more interesting for the student to demonstrate on himself the value of the muscle when using tools than to remember that its action is "abduction of the index finger from the line passing through the middle finger, and flexion of metacarpo-phalangeal joint with extension of interphalangeal joints", as most of us learned by rote.

The subject of after-care of paralysed people is not easy to discuss in lecture form. The demonstration of patients in the wards later this afternoon will help you more. I regret that the care of the largest and the most important group—the so-called "mild cases"—must be omitted today, for the care of "missed cases" and of patients with few muscles affected was discussed two years ago (Macnamara, 1950). Today I will try to record what the years have taught me in the care of severely paralysed patients, and particularly of the most difficult group—those paralysed as infants from ten days old.

I have been fortunate in living in the same place, doing the same type of work, for a long time. In twenty-seven years there has been time for seedlings and saplings I have cared for to grow into trees. There have been many opportunities to review in 1951 and 1952 patients who came under my care between 1928 and 1937, whom I have seen at intervals since. One learns more from a careful study, even of a relatively small group of severely paralysed patients cared for over two decades, than by observing hundreds of patients for a short period—up to five years.

The quality of the work of a doctor responsible for the care of a severely paralysed baby should not be judged at the end of a year or two, but at the end of the child's growing life for function and posture, and in adult life for his adjustment and his usefulness. I believe that, if the crippling following poliomyelitis is to be minimized, there should be a minimum of transfer of patients from one doctor to another, but that each doctor should regard himself as the person whose objective is to steer the patient past deformity, and to interest parents and patient in working for the minimum handicap.

The doctor must be as watchful for the first deviation from normal line of growth as he would be were the patient his own child—quick to detect and deal with such matters as an undue lengthening or shortening of the *tendo Achillis*, a radial or ulnar deviation of wrist, a shunt of the chest a little to one side of the pelvis, or the shortening of the *rectus femoris*. Every gross deformity which follows poliomyelitis, at some stage, has been a slight deviation from normal alignment.

To help in this watchful care the doctor has allies—physiotherapists and parents. Parents learn more quickly the objectives and the hazards than do members of the changing nursing staff in any large hospital. I am convinced that parental care of children in the homes, under the guidance of physiotherapists who "teach" rather than "treat", results in better attention to detail (and a consequent reduction of deformity) than can be achieved in institutions which provide long-term care for children away from their parents. We have had better results with country children during the past few years, since a plan has been implemented of keeping the child in an after-care institution until all joints are mobile; the child is accustomed to his splint, and his care is compatible with other demands on the mother. In severe cases this stage may cover the first nine months. The parent is coached in the details of care at home for the next two months. Usually ten sessions are needed. At the end of that time the patient returns for several weeks, and the parent has a second period of tuition—perhaps five sessions—in the programme for the next three-month interval at home, and so on.

Four years ago the Australian Red Cross Society, Victorian Division, established at Welfare House a hostel to which mothers of patients and patients suffering from poliomyelitis and also from cerebral palsy come at regular intervals for adjustment of appliances and instruction. This work of the Red Cross has given one solution to the problem of an alternative to the expense of long-term hospital care and has overcome the difficulty of achieving care in detail under the system of roster of nurses which has followed the introduction of the forty-hour week.

This plan of hostels for parental instruction has convinced me that the intelligence, the interest and the eye for alignment of Victorian parents has been under-estimated in the past. To develop the talents of parents it is necessary that they be taught that a splint has no mystical healing powers; that it is a gadget or contraption designed to serve specific purposes; and that adaptations are needed for growth. Parents are encouraged to handle all adjustments needed while the child is at home.

In 1867 Charles Fayette Taylor, in his book "Infantile Paralysis and its Attendant Deformities", stated categorically: "The deformities ordinarily attending Infantile

Paralysis are entirely preventable." The years have shown me that Taylor's standard is a high one; but I agree with Taylor that the virus of poliomyelitis is not directly responsible for deformity.

Deformities which follow poliomyelitis are due to the action of three factors: (i) gravity, (ii) positions habitually assumed, (iii) overaction and subsequent shortening of certain muscles, with stretching and increasing weakness of their antagonists.

While admitting that I have failed to steer some of my patients past deformity and that in a number scoliosis has developed, I think that Taylor's statement should be accepted as a standard which we should strive to attain.

If in a community there is anxiety to prevent deformity, if doctors aim at Taylor's standard, I submit that the incidence of deformity therein will be considerably lower than in one in which doctors take the attitude: "The child has had poliomyelitis. Deformities follow this disease. Let him get about as and how he can." Doctors who adopt this *laissez-faire* attitude rarely have the opportunity of seeing the end results—of judging the crippling which is evident years later. Few parents will accept for long the standard that "near enough is good enough". The patients go to orthopædists in other centres for the correction of their deformities.

When I review those of my patients of whose adult form I am ashamed, it is clear that the incidence of deformities is highest in those paralysed as babies.

During the past fifteen years, from other work—the care of children with cerebral palsy and the postural deformities of children who have had no neuron lessons—I have learnt four lessons which have altered my approach to the care of a baby paralysed by poliomyelitis. In the early years my chief objective was the care of the paralysed muscles. Now the first objective is "orthopædia"—in the sense used by André—to try to guide the patient to the form of a normal child, and especially to try to prevent or minimize scoliosis.

The four lessons are set out below.

1. Even a normal baby is susceptible, as is a cooling gelatine solution, to the position habitually assumed.

2. The normal child of good nutrition grows to a normal symmetrical posture as a result of even, symmetrical use of muscles of the four limbs and of the trunk.

3. The younger the child in whom one upper or one lower limb is weakened, the other limb being normal, the more readily does he tend mentally to "write off" the weaker limb and develop to the fullest the potential function of the normal limb. Just as the cerebral palsied child with mild hemiplegia tends to carry the affected upper limb as a "wounded flipper", so the child who has lost even slight power in the muscles of one upper limb through poliomyelitis is apt to learn to manage without it, and in so doing, distorts Nature's plan for even development of his trunk. This "mental alienation"—discarding of the limb as a useful tool—becomes a fixed habit when a child is allowed to attempt to carry on a so-called "normal" life, with one upper limb dangling because of weakness of the shoulder muscles and elbow flexors.

4. An inequality in the range of easy joint movement can, in a baby, be a cause of rapid development of deformity. For example, a baby is allowed to lie with his head resting on his left cheek while he sucks his left thumb. Rotation of his head on his trunk to the left is full, but he resents full rotation of the head to the right. When lying on his back he cheerfully turns his head to look at an object on his left on the table near his left cheek, but when attractive toys are placed on his right side he rotates his trunk, left side forward, in order to evade any increase of the range of rotation of his head to the right beyond the degree which is comfortable. This trick movement can result in scoliosis with rapid development of rotation—the long diagonal being left nipple, right scapula.

I have noted, too, that the tendency to scoliosis in little children begins within a few hours of the onset of paralysis. If deformities are to be anticipated, splinting must be applied very early and be efficient, and frequently modified.

For example, a baby whose right lower limb alone is paralysed is unable to carry out the pastime of a baby lying on his back while awake—namely, kicking first with his right lower limb, then with his left. The paralysed right limb lies on its outer border and appears to be longer, because the baby can now use only his left lower limb to kick. He does this constantly, hitching up his left (the normal) side of his pelvis, so that the left lower limb is apparently shorter. If this is not cared for, the range of flexion of the right hip joint becomes limited, and the baby, when supported to sit, will guard the right hip against full flexion. This difference in mobility accentuates the elevation of the left side of the pelvis. The pelvic obliquity results in a shorter space between the pelvis and the costal margin on the left side than on the right side. The relationship between the acetabulum and the head of the femur on the left is disturbed, so that a dislocation of his left hip becomes a real danger.

Nature's plan for the development of a potentially normal acetabulum is disturbed. The osteoblasts in the left acetabulum do not receive the stimulus necessary to lead to deposition of bone to form an adequate cover for the head of the left femur, the left thigh being adducted at the hip. The acetabulum remains shallow. Messages are not sent to the osteoblasts of the developing femur, in order to alter its form from that of the drumstick (*coxa valga*) type of the infant to the angle normally made in older children between neck and shaft.

Frequently the abdominal muscles on the same side as the affected lower limb are weaker than those on the side of the normal limb, so that the baby lies in a circle with concavity to its normal side. The stage is set for gross scoliosis and dislocation of the left hip—that is, the hip of the limb unaffected by poliomyelitis.

To deal with this, various additions to the stainless steel frame are needed: side guards to chest and pelvis; a groin strap to support the pelvis of the paralysed side; and periods of splinting in which the right (the paralysed) hip flexors are favoured, for the more quickly the stage is reached at which the baby seeks to resume his even alternate use of lower limbs when free to move, the sooner the danger of an unnecessary handicap, a dislocation of the hip of his good side, is averted.

Because he grows quickly, splinting needs modification at short intervals—six to eight weeks. The older patient who had walked for years before he developed poliomyelitis, when fitted with a brace to support a weakened lower limb, will try to cooperate in standing evenly. The baby with similar weakness of one lower limb, paralysed before he has attained standing balance, needs months of education in the "feel" of two limbs equally stable.

For this reason we have, within six to twelve months of the onset, depending on the age and on the correction of the pelvic hitch as shown by X-ray examination, supplied little children with a standing splint, right and left leg braces connected with a trunk support with pelvic and chest bands. No joint is supplied to allow hip flexion. The splint is used only for standing.

The degree of abduction is determined by the X-ray demonstration of the alignment of the developing femora and their relationship with the pelvis.

By this means the child can pass some hours each day in a standing position. Later, the trunk extension on the splint is reduced to a pelvic grip, and later, if the spine is straight, if the pelvis is true, and if the affected limb still requires it, only a single brace is used. The relationship of spine, pelvis and each femur is checked by X-ray examination at regular intervals.

Similarly, the child with weakness of the muscles of one upper limb demands just as much care to guide his growing trunk in straight alignment as he requires measures to encourage return of power to the affected muscles. For this reason all children whose shoulder muscles are involved are supplied at the onset with a frame to keep the trunk straight while the shoulder muscles are protected.

When it is obvious that the trunk and lower limbs have escaped or recovered, an ambulatory deltoid splint is obtained, its purchase being taken from a band tailored to the pelvis. Sitting without the ambulatory splint is allowed when a chair and table are provided to fit him exactly; the former has a foot rest and pelvic and chest guards to prevent any shunt of the chest lateral to the pelvis; the latter is high enough to support both elbows and forearms when the shoulders are abducted to 70°. Often a guard is needed behind the dorsal aspect of the affected arm to prevent shoulder extension.

The periods of use of the chair and table and of the ambulatory splint are cautiously increased, the child spending the remaining hours in his frame. When full freedom is allowed in the day-time, the frame is used at night only for a long period. Many fathers make or modify these chairs and tables. The parents see for themselves that an asymmetrical ambulatory deltoid splint should not be worn for long periods each day, for use of the arms is uneven. On the raised table, given support, the weaker arm has an opportunity for use equal to that of the normal arm, if the muscles below the shoulder have escaped or recovered.

The years have taught me that one should not concentrate only on the care of the paralysed muscles, but view the patient as a whole. For this reason, while agreeing that no careful assessment of progress can be made unless muscle charts are kept, I think it is important for the doctor responsible to look at the patient frequently and carefully and to consult the muscle chart at relatively long intervals, or when it is necessary, to give accurate data to students of the patient's condition when care commenced.

Sometimes it is obvious within six or twelve months of the onset that the paralysis of the lower limbs is so severe that crutches or sticks will be necessary. One objective then is the development of superlative power in the upper limbs and trunk. A person with little power in the muscles of the lower limbs, wearing braces, can negotiate steps with the help of sticks if the muscles of the trunk and upper limbs are stronger than normal.

Sometimes the degree of recovery in an upper limb is the factor which determines when efforts should be made to acquire standing balance as a preliminary to walking. If in a patient both lower limbs are severely paralysed, while muscles of an upper limb are involved to a degree which promises complete recovery, any attempt at walking which involves the use of crutches, sticks or a brace should be postponed until the affected muscles of the upper limb have recovered the maximum power, and the endurance which makes it safe for them to sustain the efforts involved in using sticks or crutches.

It is important that every paralysed person, as soon after the subsidence of acute symptoms as possible, should be turned into the prone position each day. Mr. Roland McEwen has shown how this procedure reduces the tendency for stasis in the pelvis of the kidney and lessens the accumulation of "renal mud", the precursor of kidney stones. The provision of apparatus which allows periods of standing, before the lower limbs, if unsupported, are capable of taking weight evenly without deformity, affords additional opportunities for kidney drainage.

Many factors influence the planning of the *régime* of care for each individual patient. For example, the problems presented by a paralysed adult, the support of his family, are very different from those encountered in caring for a baby paralysed at five weeks, even though the same muscle groups have been affected and to the same degree.

The man has a solid bony framework. His acetabula are formed; each femur has a head, an angle and a neck. He runs little risk of dislocating the head of the femur on the unaffected side, for his habits are different. Alternate kicking of the lower limbs when awake and lying supine is not his pastime. The older patient in whom growth is complete does not demand so frequently the vigilant checking of apparatus as is required by young children.

On the other hand, the limitation of spinal flexion and the hamstring tightness which remain after paralysis as a persistence of the "spine sign" which characterized the pre-paralytic stage, give more trouble in older patients than in babies.

By the use of saline baths, posturing and frequent passive movements the normal range of movements of joints should be restored and care taken that muscles which pass over more than one joint become their normal length—the hamstrings, the *rectus femoris*, the flexors of the wrist and fingers.

The little child has no worries, no duties to other people. The paralysed adult has many anxieties in addition to his paralysed muscles, and for many months may be a difficult, sick, miserable person.

When this stage is over and nutrition and general health are good, progress is made. The adult quickly learns the objectives and the principles involved, and he grasps the economic importance of the function of various muscle groups. Sometimes one has to help him to a compromise, sacrificing the optimum opportunity for his recovery to the plans for his career. Before such a decision is made he should be educated to be able to evaluate the alternatives—the hazards and long-term handicaps which will follow early attempts to resume activity even with the support of appliances, or the prospect or certainty of useful return of power if activity is postponed and protection continued.

The adult who develops poliomyelitis runs no risk of unequal growth of lower limbs. The difference in temperature between a normal and a severely paralysed limb of a baby can be appreciated even in warm weather. By means of woollen covers, hot-water bags *et cetera* the temperature can artificially be kept even while the child is recumbent and disparity in growth delayed.

When activity is allowed, the growth of the normal limb, stimulated to take more than its share of work, may exceed that of the paralysed limb. Last year Dr. A. Hamilton, of Sydney, at a meeting of the Australian Orthopædic Association asked members who are caring for these patients to apply a tourniquet to the weaker limb three times daily for a period up to twelve minutes, firmly enough to cause venous congestion. It is too early to judge whether Dr. Hamilton's revival of this old plan, "hyperæmic treatment", will lessen the disparity in growth between the two limbs; but my impression is that it is worth while continuing for the long period needed to acquire data.

Recently there has arisen a school of thought which questions the utility of any splinting in the after-care of poliomyelitis and a tendency to attribute this idea to Miss Elizabeth Kenny's teaching and influence. This seems to me quite unfair to Miss Kenny. When sufficient time has elapsed to evaluate Miss Kenny's contribution to poliomyelitis and to paralysed people I consider it will be agreed that it is covered by five headings: (i) The first, and perhaps the greatest, is that Miss Kenny has been able to use the power of the Press as no doctor would have been allowed to do, to make governments and public realize that an epidemic of poliomyelitis calls for the expenditure of public money. The provision of after-care to Victorian poliomyelitis patients has been extended because money has been made available. For the lay appreciation of the fact that many poliomyelitis patients recover, that provision of care is imperative, all paralysed people and those caring for them are greatly indebted to Miss Kenny. (ii) Miss Kenny provided a stimulus to research workers to abandon the servile adherence to the theory of essential neurotropism of the virus of poliomyelitis and swing back to the conception which many clinicians held over all the sterile thirties—namely, that the death or even the involvement of nerve cells is only a part of the pathogenesis of poliomyelitis. (iii) Miss Kenny has protected weakened lower limbs from weight-bearing for long periods. She has endeavoured to hold trunk and limbs in comfortable positions compatible with ultimate good posture. She has used many contraptions which, though not of metal, may fairly be called splints. Miss Kenny has employed a staff large enough to police the position of the patient and of his limbs. We have used more robot police staff called splints to attain the same objective. (iv) Miss Kenny, with justification, has drawn attention to the harm which

follows the practice of confining patients in splints in institutions which, through staff difficulties, could not arrange for movements to be carried out at intervals short enough to ensure first attainment and then maintenance of normal range of joint movements. She has stimulated me to try to develop further the care of paralysed people in homes, for in the homes this difficulty does not arise. (v) Miss Kenny has stressed the harm which can be done by careless splinting, by the use of poorly designed and ill-fitting splints; a fact stressed by Taylor eighty-five years ago. Modern technology has given us some new materials, but the efficacy of any splint still depends on the experience and vigilance of the doctor.

Splinting may be carried out perfunctorily; a brief order may be sent to a metal worker, all the details which are essential for his intelligent cooperation being omitted; the splint-maker or the parents may be allowed to apply the splint without checking by the doctor of its alignment, or without coaching of the parents in its purpose and in details of its application.

Such "lip service" to the use of splints can readily result in trouble. However, it would appear from Miss Kenny's use of the term "orthodox treatment" that she did not have the opportunity of learning the wide variation in standards. There are varying standards in cooking—careless, haphazard cooks and others who are prepared to take infinite trouble to prepare food well. The fact that some food is spoiled by careless cooking does not justify a gospel that all food should be eaten raw. I consider that accurate, evaluated splinting is our most useful tool in the prevention and reduction of crippling following poliomyelitis.

This conclusion rests on three basic facts: (i) Any muscle, normal or paresed, is at a disadvantage and becomes weaker when continually stretched. Conversely, it is favoured and later becomes shortened when its origin and insertion are continually approximated. (ii) The position habitually assumed is one factor which influences the line of growth of any growing thing, be it plant, animal or child. (iii) The law of gravity must be remembered.

To illustrate the effect of gravity, consider a patient whose paralysis, two weeks after the onset, is limited to the flexors of the right elbow. Paralysis is not complete. The elbow can be flexed voluntarily when gravity is eliminated, the arm and forearm and hand being supported on a high table; but the patient is unable to lift an object grasped in the strong right hand. The limb is allowed to drop. The weight of the forearm and hand stretch the elbow flexors. The ability to flex the elbow, on the level, disappears. After an interval the patient notes that he has lost this movement and he has come to appreciate the reduction in the economic usefulness of a good right hand, which results as a loss of elbow flexion against gravity. He seeks help. The flexors of the elbow are then constantly protected. The forearm is supported. Extension of the joint is limited to movements on the high table only as often as is necessary to keep the range of joint movement easy and painless. After an interval not so long as that in which the weakened muscles were insulted by stretching, the elbow flexors have regained the power to flex the elbow on the level. They have been brought back to the stage from which, as Taylor bluntly said, "they should not have been allowed to depart". Gradually, with continued protection, they gain power against gravity, later against gravity and resistance, until the aim of an adult woman patient is achieved—"I can lift a saucepan!"

I have watched this sequence many times in many muscle groups—the demonstration that a muscle whose power, obvious within a few weeks of the onset, has disappeared because of stretching, can store the capacity to regain power and to recover even after long intervals if protection from stretching is arranged.

There are many such instances in the writings of Sir Robert Jones and of Taylor—workers whose interest in the behaviour of paralysed muscles was long continued.

The word "care" has been used throughout this paper as opposed to "treatment". It implies more clearly than does treatment that recovery of muscles paralysed by poliomyelitis is Nature's work. As I see it, our place is to assist the natural tendency to recovery and to provide protection from the factors which defeat her and deform the patient—namely, fatigue of muscle, muscle imbalance, gravity, and deforming positions habitually assumed.

It would be ironical if Miss Kenny's real contribution to the care of paralysed people was negated in some centres by twisting of her teachings to sponsor a gospel of neglect.

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PUBLIC HEALTH ASPECTS OF POLIOMYELITIS IN VICTORIA.[1]

By BERTRAM P. MCCLOSKEY, M.D. (Melbourne),
Melbourne.

POLIOMYELITIS has always been considered a public health problem in Victoria, but in recent years the activities of the Department of Health have been extended to provide a new comprehensive service to the community. The Victorian policy in regard to the poliomyelitis services is determined by the Consultative Council on Poliomyelitis, which functions as an advisory body to the Minister of Health. It is appointed by him and consists of nominees of the British Medical Association, the Masseurs' Registration Board, the Children's Hospital, the Australian Physiotherapy Association, the Medical Superintendent of Fairfield Hospital, the Victorian Division of the Red Cross Society and the Victorian Society for Crippled Children.

On the recommendation of the Consultative Council a poliomyelitis division has been established in the Department of Health, and the following services are provided: (i) An epidemiological investigation of reported cases of poliomyelitis by departmental medical officers. (ii) A diagnostic consultant service. (iii) After-care treatment facilities. (iv) Public health measures.

Epidemiological Investigation of Reported Cases.

Standardized investigation forms are being used to record data which will be subsequently transferred to punch cards for analysis. Practitioners have been asked to cooperate by promptly reporting cases to the department, using the telephone where practicable, in addition to notification in the usual way.

Diagnostic Consultant Service.

The department retains the services of a panel of physicians for the diagnosis of doubtful cases. In the metropolitan area the practitioner communicates directly with the consultant of his choice, and elsewhere in the State regional consultants are available. In special circumstances country practitioners may obtain the services of a metropolitan consultant by telephoning the Chief Health Officer, who has the power to authorize such a consultation.

After-Care Treatment Facilities.

Metropolitan Area.

The following after-care treatment facilities are provided in the metropolitan area.

[1] Based on a talk given as part of a symposium on poliomyelitis arranged by the Melbourne Permanent Post-Graduate Committee in conjunction with the Consultative Council on Poliomyelitis held on March 29, 1952, at Fairfield Hospital.

Medical Treatment.—The services of departmental medical officers specially trained in after-care treatment are available to practitioners desiring orthopædic supervision of poliomyelitis patients over fourteen years of age unable to afford private consultant advice. (Similar patients under fourteen years of age can receive treatment at the Children's Hospital.)

Physiotherapy.—The services of departmental physiotherapists are available for the physiotherapy treatment of poliomyelitis patients over fourteen years of age, irrespective of the financial status of the patient, provided the patient is not attending a public hospital for treatment. This service is available through medical practitioners only and not directly to patients. The departmental physiotherapists also provide physiotherapy to patients in the poliomyelitis unit at Fairfield Hospital, to the Red Cross hostels known as Welfare House and Rockingham, and to the poliomyelitis unit at the Commonwealth Immigration Centre at Somers.

Country Areas.

The following after-care treatment facilities are available in country areas.

Medical Treatment.—Poliomyelitis patients are regularly reviewed by orthopædic specialists at clinics conducted in most of the large centres of population. Usually these clinics are held at base hospitals for both in-patients and out-patients, their frequency being determined by the number of cases requiring review.

Physiotherapy.—In most country centres physiotherapy is provided by the base hospital physiotherapy staff, but departmental physiotherapists provide domiciliary treatment in the Geelong, Warrnambool, Ballarat, North Eastern and Gippsland areas. This service will be extended when more trained physiotherapists are available. The department places cars at the disposal of the physiotherapists so that their services may be fully utilized.

Splints and Appliances.

The department, by arrangement with the splint shops of the Royal Melbourne and Children's Hospitals, supplies splints and appliances when ordered by the patient's orthopædic consultant.

Two hundred perambulators owned by the Department are on loan to patients with poliomyelitis. No charge is made for their use.

A stock of 200 "Foster" type universal splints is held by the department in readiness for use in the next epidemic to minimize the delay usually experienced in epidemic times between the ordering and the receiving of splints.

Transport of Patients During Convalescence.

Ambulance or hire-car transport is provided on approval by the Minister when required—except for interhospital transfers.

Provision of Respirators.

The department is responsible for maintaining an adequate supply of respirators in Victoria. In addition to respirators on loan to hospitals in all the main centres of population, the department holds a pool of seven respirators. Respirators can be sent immediately to any hospital requiring them.

Nursing Services.

The department retains the services of three visiting nurses for the after-care of poliomyelitis patients.

Research.

Departmental officers cooperate with research workers in the collection of material for study. At present Dr. A. Ferris, of Fairfield Hospital, Dr. K. Bowden, of the City Coroner's Office, and Dr. P. L. Bazeley, of the Commonwealth Serum Laboratories, receive specimens. Specimens are stored in a suitable form, so that there will be an adequate supply of material for study when the facilities are available for typing strains of virus isolated from stools.

Public Health Measures.

The department periodically circularizes the profession regarding measures recommended to limit the spread of poliomyelitis and sets out precautions which should be taken to prevent the onset of paralysis in what would otherwise be abortive or non-paralytic cases.

Measures to Limit the Spread of Poliomyelitis.

It is realized that the measures recommended are not sufficient to prevent the epidemic spread of poliomyelitis, particularly in view of the large number of subclinical infections with the virus. Nevertheless, the measures aimed at checking the spread of poliomyelitic infections are considered justifiable for the following reasons:

1. By isolation of patients suffering from clinical poliomyelitis the spread of infection may be delayed, since there is evidence that such children may excrete virus in greater quantity and for a longer period than do those with abortive infections.

2. The probability of recovering virus from contacts has been shown to depend on the intimacy of contact. Isolation of intimate contacts of an infected patient may well reduce the total amount of virus distributed in the community. Measures aimed at deferment of infection till a later age are justified, since it is at least possible that a prophylactic vaccine may be developed in the near future.

3. Lastly, public opinion demands that the authorities should do what is possible to prevent extension of epidemics from place to place, even though such measures may fall short of their objective.

The following measures are recommended to limit the spread of poliomyelitis.

Isolation of Contacts.—The Infectious Diseases Regulations of Victoria direct as follows:

> All contacts attending school and all contacts under school age to be isolated for a period of at least twenty-one days after the commencement of the patient's illness; and
>
> All contacts to be kept under surveillance by the medical officer of health for such period of time as he considers necessary.

Strict enforcement of this regulation is advised.

Prevention of Food Handlers Who are Contacts from Handling Food.—Under the Infectious Diseases Regulations there is no provision for the isolation of adult contacts or for their exclusion from their occupation. In the case of food handlers it is desirable that they should be excluded from work. The Infectious Diseases Regulations provide that "all contacts be kept under surveillance by the medical officer of health for such period of time as he considers necessary". It is recommended that all food handlers be placed under strict medical surveillance and be asked to report daily. In the event of any signs of illness they should be isolated as suspect sufferers from poliomyelitis until such time as poliomyelitis is excluded, or alternatively for twenty-one days from the date of onset of symptoms.

Restriction of Mass Movements of Children and Widening of Spheres of Contact.—During the recent epidemic the Consultative Council stressed the need for the restriction on the movement of children to and from areas in which cases had been notified and recommended the cancellation of all gatherings such as sports meetings *et cetera* in such areas. The Education Department cooperated in giving effect to this recommendation.

Personal Advice on Hygiene: Drinking Facilities at Schools.—On the Council's recommendation a circular was distributed to the headmasters of all private and State schools giving advice with regard to the personal hygiene of children during the poliomyelitis epidemic, particularly concerning the use of common drinking taps, hand towels *et cetera*. Over the signature of the members of the Consultative Council a statement was also issued to the public containing advice regarding hygiene.

Closure of Kindergartens and Schools: Transfers from Schools and Crèches.—During the recent epidemic the Consultative Council recommended that a kindergarten be immediately closed whenever a case appeared in it. This advice was implemented with the cooperation and assistance of the Maternal and Child Hygiene Branch of the Health Department. The Council did not recommend the closure of schools, as it was considered that the provisions of the Infectious Diseases Regulations, which provide for the exclusion of all child contacts for a period of three weeks, sufficiently met the need in this regard. On the advice of the Consultative Council the Education Department refused to admit transferred scholars for a period of three weeks if the scholars in question had come from an area where cases of poliomyelitis had been notified. Crèches were not closed when any case was notified from the children attending, as this would have meant considerable hardship to the mothers, most of whom leave their children at the crèches so that they can go to work.

Swimming Pools.—The Council did not recommend the general closure of swimming pools, but advised that attendances should be strictly limited, and the managements were requested to ensure that no overcrowding occurred. In their advice the Council pointed out to the controlling authorities that chlorination of water did not immediately kill the virus of poliomyelitis.

Precautions to Prevent the Onset of Paralysis.

In addition to the above measures, the following precautions are advised to prevent the onset of paralysis in persons infected with poliomyelitis virus.

Tonsillectomy.—The Consultative Council recommends that in times of high poliomyelitis incidence in any district the medical practitioner should discuss with the parents or guardian of the patient the relative risk of bulbar poliomyelitis following tonsillectomy and that of deferring operation until such time as the poliomyelitis incidence is low.

Inoculations.—The Consultative Council recommends that during periods of high poliomyelitis incidence all inoculations should be given subcutaneously when practicable and that these should be reduced to a minimum in children over six months of age. Particular emphasis is placed on discontinuing pertussis vaccine inoculations whether alone or combined with diphtheria toxoid. Attention is drawn to the need for adequate syringe and needle sterilization. In all cases syringes and needles should be sterilized by boiling or autoclaving; in no cases should reliance be placed on chemical sterilization, which is not considered effective for syringe or needle. For skin sterilization, spirit by itself should not be used as it is inefficient; detergents in spirit, such as tincture of "Zephiran" or "Cetavlon", are recommended. The policy of the Department of Health in relation to immunization procedures has recently been revised, partly to offset the risk of poliomyelitis following immunization.

Exertion.—Because physical activity of the patient on the day of onset of the meningitic phase has been shown to influence the severity of the paralysis, the Consultative Council recommended during the recent epidemic that all children with a febrile illness should be put to bed and given complete rest for at least three days.

Transport of Patients During the Acute Stage.—As it has been shown that paralysis is severer and the mortality rate higher in patients transported long distances during the acute stage, the Consultative Council issued a circular to all medical practitioners in Victoria drawing attention to the risks of movement of the patient in acute cases of poliomyelitis.

Pregnancy.—As the risk of death in pregnant women who develop poliomyelitis during the third trimester is considerably greater than that of those developing poliomyelitis during the first or second trimester, it is recommended that ante-natal visits be arranged so that pregnant women do not sit in waiting rooms with other patients, and that strict precautions regarding sterility be taken when pelvic examinations are performed, particularly during the latter half of pregnancy.

Dental Extractions.—The following advice was issued by the Consultative Council to the School Dental Service:

> Tooth extractions should be limited to cases of immediate necessity during the epidemic; further
>
> Dentists and attendants should wear masks or deflectors and pay special attention to the sterilization of anything animate or inanimate coming in contact with the patient's mouth or mouth secretions. The wearing of sterilized gloves by dentists is suggested.

Comment.—The above factors have been selected from the host of factors known to influence the onset of paralysis in cases of poliomyelitis, as they are of immediate public health importance.

Conclusion.

In conclusion it should be stressed that paralytic poliomyelitis is one of the most important public health problems in Victoria. Although the control of poliomyelitis is not yet possible, control of the onset of paralysis is possible in many individuals developing poliomyelitic infection. In most cases in which paralysis develops the effects can be minimized by prompt and effective treatment as outlined by Dr. H. McLorinan and Dr. Jean Macnamara in this symposium.

The Consultative Council on Poliomyelitis in Victoria through the Minister of Health is ensuring by the provision of adequate facilities that until such times as effective measures are available to control poliomyelitis its effects in the community will be minimal.

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THE POLIOMYELITIS EPIDEMIC OF 1950–1951 IN THE NEWCASTLE AREA.

By ETHEL BYRNE, M.R.A.C.P., and ALAN T. ROBERTS, F.R.C.S. (Edinburgh),
Newcastle.

THE poliomyelitis epidemic commenced in the city and suburbs of Newcastle in October, 1950. There was a pronounced decrease in the number of cases at the end of December, 1950, and for two weeks there were very few cases. A recrudescence of the epidemic occurred and persisted for the next three months, January to March, 1951. This was characterized by a predominance of mild cases, but a much greater percentage of deaths from bulbar paralysis. In 1950 there were 115 cases and 10 deaths, and in 1951 50 cases and 7 deaths.

The population of Newcastle and its adjacent towns is 180,000, and practically every patient in that area was treated in this clinic. There were in addition 85 cases from outlying country districts. The total population of these country towns is slightly less than that of the Newcastle area. The percentage of deaths and the percentage incidence of disability paralysis were higher in these cases as compared with the Newcastle area. It is presumed from this that those 85 cases represented a much greater number of affected persons and that only the more seriously ill were sent away from their home town.

There were in all 250 cases; 66% came from Newcastle and suburbs and 34% from outlying country districts.

Bulbar Poliomyelitis.

There were 50 cases of bulbar poliomyelitis and 30 deaths. We followed the lead of James L. Wilson and used this term for "the type of disease where the medulla is attacked, resulting in paralysis of one or more of the muscles innervated from that area, such as the pharynx, soft palate, larynx, facial muscles, or disturbances in the respiratory and vasomotor centres, ill defined in position though they may be. The term 'bulbar poliomyelitis' does

not refer to respiratory muscle paralysis, i.e. involvement of the diaphragm and intercostal muscles which are innervated from the cord."

We did not find the use of respirators to be of any assistance in our cases of bulbar poliomyelitis, and we do not believe that their use can relieve conditions resulting from involvement of the brain stem, which produce respiratory abnormalities by other means than respiratory muscle paralysis. These patients were not cyanosed.

Of the 30 patients who died, 22 were submitted to post-mortem examination and the findings were remarkably uniform, comprising gross involvement of the brain stem and spinal cord. We had no patients of the other group—that is, with involvement of the diaphragm and intercostal muscles—for whom a respirator might be considered necessary. The surviving patients with bulbar paralysis, the majority of whom were seriously ill at the time and needed intensive drainage and suction therapy, recovered quickly and were all discharged from hospital within six weeks. When 50 non-paralytics and 50 patients with bulbar paralysis are omitted, there were 150 with the spinal paralytic type of poliomyelitis with sufficient evidence of paralysis to warrant their admission to hospital—60% from the Newcastle area and 40% from the outlying country districts.

Management.

All patients were admitted to hospital under the physician, and the orthopædic surgeon had contact with them as soon as the diagnosis was confirmed. Routine lumbar puncture was not performed; lumbar puncture was performed only when the physician in charge considered that the condition might be some other disease, in which the result of the lumbar puncture might have an influence on the treatment.

A bulletin was issued to the relatives twice a week. This gave the duration of the patient's stay in isolation and probable future stay in the orthopædic hospital, the area affected and the probable future outlook for disability. Copies of this bulletin were available at several sources of inquiry, and relatives were able to obtain information readily. In previous epidemics it had been a constant source of discontent that no reliable information was readily available. This procedure was found to be of great benefit to both relatives and the attending doctors. The relatives of all dangerously ill patients were, of course, kept more closely informed. Subsequent reference to these bulletins revealed a close degree of accuracy in forecasting the future disability, except in the case of infants and those with widespread apparent paralysis.

Treatment in the acute stage consisted of rest in bed without the use of any restraining splints, daily hot baths as soon as the temperature was normal, and hot fomentations applied to as many patients as possible, especially those in whom the muscular spasm and pain were pronounced symptoms. All patients were encouraged to move and turn about in bed, and were allowed to get up after the three weeks' period, or beforehand if their condition warranted it. The only immobilization used was in the form of pillows to posture the patient with comforting support and to prevent any completely paralysed limb from remaining persistently in an abnormal position. Every attempt was made to put the joints through a full range of movements several times a day, and stretching of the painful muscle groups—for example, calf and hamstring groups—was carried out daily in the bath. The extent to which this was done was regulated by the amount of pain which was present, and it was not conducted in a forcible manner.

On the termination of the three weeks' period of isolation a large percentage of the patients were discharged to their homes and instructions were given to the parents for the maintenance of full range of movement in the joints.

The type of patients discharged from hospital were as follows: (i) Nearly all those with upper limb paralyses, only those severely affected being kept in hospital. (ii) Patients who had no residual paralysis, but who were not completely normal owing to some remaining muscle stiffness. Subsequent follow-up examination showed that it would have been wiser to transfer patients of this type to the orthopædic block for one or two weeks, where their activity would have been better guided than at home. Ultimately their discharge from hospital did not affect their cure, but it was retarded for some weeks, as the parents were fearful of allowing them to move about and had not had the opportunity of learning how to conduct their treatment. (iii) Most infants whose home conditions appeared to warrant their effective supervision by the parents. We do not think it is possible to reeducate infants other than by playing with them. The parents, particularly when instructed what to do, have more opportunity of doing this than a busy physiotherapy department.

The balance of the patients were transferred to the orthopædic block of 40 beds, which was available for the epidemic. At no time was lack of accommodation an embarrassment, and we had no need to discharge patients from hospital earlier than we might want to owing to shortage of beds.

At the commencement of the epidemic the nursing service only was available, and nurses were not specially trained in physiotherapy and were apt to be changed frequently to other wards; but with the cooperation of the nursing administration several nurses who had shown an aptitude for the work remained and soon acquired proficiency.

We were fortunate in obtaining the services of a trained physiotherapist, who had not been practising for some years and was receptive to the ideas prevailing in the clinic. Although she was able to work only part-time, she was able to initiate treatment and teach its principles to the nursing staff, and in a short time all patients were receiving full and efficient treatment. It was amazing to see the influence that one skilled person could have on the conduct of the treatment. In our opinion the most important factor in the success of our methods was the ability of this physiotherapist to secure the cooperation of the patients in our campaign for activity.

We do not state that treatment effects a cure or a recovery, and we would agree with Steindler's statement made in 1946, when he was discussing treatment: "Because of the high incidence of spontaneous recovery any therapeutic measure would be hard to evaluate." But we also agree with his statement that "we cannot be blind to the fact that in many perplexing problems concerning anterior poliomyelitis, experimental medicine and basic medicine have opened our eyes to certain traditional mistakes and misconceptions which will have to be corrected or revised".

Physiotherapy.

In the outlining of a basis for the reeducation of these patients, following on their initial acute stage, the principle was adopted of first trying to obtain general movement if we were unable to elicit a response from any single muscle or group. Routine individual muscle charting was not carried out, but the progress of the groups of muscles was recorded. Our only trained physiotherapist had not the time to do this work, and we considered that information obtained by other assistants would not be sufficiently reliable to record and use as a basis for estimating the value or otherwise of the treatment.

One great advantage of treatment in the water bath is that the attending doctor has the opportunity of checking the muscle progress of ten to twelve patients at one visit while they are exercising in the bath. The absence of splinting also facilitates the conduct of frequent examinations.

No movements were discouraged, and trick movements were developed if there was no response to the normal movement. The function of the limb as a whole was the main objective. In the lower limb the importance was stressed of establishing the stability of the hip, knee and ankle joints in that order. An efficient foot is of very little use unless there is stability of the knee and hip joints. In the upper limb the return of function to the hand and fingers would be of great value to the patient in his usefulness, even if he failed to regain abduction of the upper arm. We observed no cases of muscle fatigue in the affected muscles, and very close observation was made on this subject. There was no instance of a muscle which

had shown an initial response displaying a tendency to lose power as the exercises were maintained.

We do not regard the grading of muscle charting as being the only factor in the ultimate efficiency of a limb. We were struck by the fact that certain patients whose muscles gave practically no response to testing were able to obtain stability and the use of their limbs and to become active without the use of any supports. In past years it has been our observation that in long-standing cases the patients having sometimes been received from other centres, activity and usefulness could be much increased by encouragement and general exercises, but the muscle chart records did not alter in any way.

A rather remarkable case came under review recently.

The patient is now aged twenty-eight years and she had an attack of poliomyelitis at the age of two years. Six years later her ankle was stabilized in equinus by an astragalectomy. She went through her school years, played games and rode a bicycle, is now married with one child, does her housework, and walks half a mile to the railway station. She walks with a limp, as the equinus position does not compensate for all her shortening. She has a stable knee-joint without recurvation when walking. The only muscle power demonstrable in her lower limb is a slight power in the quadriceps, which enables her to agitate the leg through a few degrees of movement when sitting on the edge of the couch. There is no demonstrable power in the hamstrings, but her psoas muscle is active and her gluteal muscles are partially so. For twenty-six years she has been able to use the leg and to keep the knee stable without the presence of muscular power and without wearing any support.

The usual practice in an extreme case such as this would undoubtedly have been to fit a caliper at an early age and the patient would never have had a chance to gain this stability.

We consider that most children, and especially those in country districts, have a happier and more active life up to their adolescent years without a restricting caliper on one leg. They can ride horses and bicycles to school, go swimming without the added trouble of removing an appliance, and even use crutches for extra activity. When the time comes for them to enter some occupation which entails prolonged standing, we fit them with a caliper from which they receive great benefit, but they have not lost the capacity for active enjoyment in their leisure hours.

Reeducation was directed on the basis of encouraging and stimulating every form of movement and activity. No patients remained in bed or received treatment in bed. Every patient was up and proceeded to the treatment, dining or school room in some way or other—wheel-chairs for those most affected, pushed along in some instances by others who were not sufficiently stable to walk without support. Tricycles and walkers were used extensively. Underwater treatment was much used. There were two large baths capable of taking six to eight small children at one time. The community and competitive spirit was developed to the utmost, and the general atmosphere of the hospital and treatment room was cheerful and bright. Nobody was hindered from exercise in any way whatever, except in forced rest periods during the day. Russell states that "little experiments in methods of treatment in the chronic stage, unlike the acute, are perfectly justifiable and within limits should be encouraged", but questions the value of prolonged rest in the neutral position. He asks: "What is it hoping to achieve? Does it achieve it? Is anything lost by early freedom of movement under supervision? . . . Can the strength of the trunk muscles be accurately gauged by our clinical methods of testing them while the patient is in bed? . . . Is fatigue harmful and, if so, how is it best avoided?" We have been asking ourselves these questions for many years and have continued to alter our methods of treatment as a result of our observations.

Seddon makes a strong plea for a realistic approach to the problem and a simplification of treatment:

> Recovery from poliomyelitis is a spontaneous affair . . . . The return of useful voluntary power is usually over within 6-8 months from the date of onset . . . . Early movements are not harmful and there seems no justification for treating affected muscles with peculiar tenderness and resting them for prolonged periods as has been done in the past with disastrous consequences . . . . The most useful forms of exercise are those performed with slings and springs and in a water bath . . . . After eight months, treatment, if necessary, should be directed entirely to securing the greatest possible hypertrophy of remaining muscles as in athletic training.

Many years ago, in the splinting era, we observed that the restless children, who were wriggling and trying to regain activity in spite of their splints, always made a good recovery in comparison with those who took their immobilization placidly. We were much impressed by the principles of Miss Elizabeth Kenny's treatment and quickly absorbed Miss Kenny's clinic into the hospital's physiotherapy department. We have not found it practicable to carry out her ideas throughout, but we feel that her original ideas are responsible for the more rational and simple outlook on treatment which is being shown in other countries, and which has not yet manifested itself to any extent in the capital cities of Australia.

Comment.

Encouragement of the general morale and the desire to move and regain activity are most important factors in the rehabilitation of those who are suffering from the after-effects of poliomyelitis. We consider that the practice of depriving individuals of their activity, limited as it may be, and of forcing them into a state of inactivity, except when they are allowed to move at rare intervals (intervals which are growing rarer as the shortage of physiotherapists is becoming greater), is calculated to produce and develop a state of inertia, which has a very harmful effect on their general outlook towards recovery. This could be tolerated only if there was adequate and positive proof that they would gain some incalculable benefit from these procedures. A percentage increase in their muscle power is presumed to be the result, but this presumption is now strongly under challenge, quite apart from the grave psychological damage that may be done.

In comparison with previous epidemics we consider that our total disabilities are much fewer in this one. These results have been achieved with a minimum of hospital accommodation and an absolute minimum of trained assistance. Regular follow-up clinics are conducted and will be continued for some years. No patients are attending for out-patient physiotherapy. We do our utmost to teach the parents and the patients themselves what they have to do and in what way they must exercise, and the results are most gratifying. If we find that the patients or their parents are neglecting their instructions, we readmit the patient to hospital for a few weeks. We have had to do this on only three occasions. A few have fallen by the wayside. Their parents will take no interest and will not bring them in to the follow-up clinics. This, we think, would be inevitable in any system except that of keeping them in hospital for years. Even then the after-care in the development of posture and rehabilitation must be associated with the maintenance of interest by the patients or their parents. In these times long-continued out-patient therapy is too disturbing to the modern way of life to be efficient. Normal activities, school *et cetera* are interfered with, the patient (or his parents) eventually tires, and he is then left without any plan of conducting his own treatment because he has been taught that it is essential for a specially trained person to control his exercise.

Our review clinics are held at intervals of one, two or three months, according to the nature of the case and the distance that the patients must travel to attend, as a large number of them come from outlying country districts.

Certain factors in our set-up were to our advantage. The first of these was continuity; the two medical officers had been in charge of poliomyelitis patients for the past thirty years. The matron of the Infectious Diseases Hospital had had the management of four previous epidemics. The control of physiotherapy was in the hands of a therapist who had ceased to practise fifteen years ago and was able to approach the problem of treatment with an open mind.

All these people are satisfied that the patients in this epidemic improved more rapidly and regained their muscular power in a shorter time.

General adoption of these simple methods of treatment would make it possible for country centres and suburban hospitals to undertake the treatment of their own patients with the assistance of a few trained physiotherapists and a medical officer, who would visit the areas and teach the available staff how to conduct their own centres of treatment. We think it would be of greater benefit to humanity to rush a water bath rather than a respirator to the scene of an outbreak of poliomyelitis. Every attempt is made to impress on the patient, during the convalescent stage and later in the rehabilitation stage, an interest in future employment and a useful life in the community.

In making an analysis of these cases for the assessment of disabilities, an attempt has been made to maintain the same principle that was adopted in treatment—namely, the future place of the patients in the community. We have divided them into three categories: (*a*) Those who will not have a disability which will prejudice their chance of employment or will hinder them from entering any sphere of work which they may desire to take up, except the most specialized. (*b*) Those whose disability entails selected occupation. All who have to wear a brace or artificial support would come into this category, but not necessarily those who are left with a weakened individual muscle—for example, triceps, deltoid, gastrocnemius or abdominal group. Most psoas weaknesses would come into the category in the case of men, but not necessarily in women. (*c*) Those who would be candidates for an invalid pension. Those with two calipers or instability of the trunk muscles with other weaknesses are considered to be in this group. (It may be that certain patients will have the mental capacity to live a useful life in the community in spite of great physical disability.)

In the Newcastle area alone the effect of this epidemic on this community of 180,000 persons was as follows. There were 17 deaths—an incidence of about 1 in 10,000. Three people were badly affected and will have difficulty in finding employment. They can all get about and are not bedridden. Twelve people will have to seek selective employment.

In the surrounding country areas there were 13 deaths. Two people were badly affected and will be in the invalid class. Eight people will have to seek selective employment.

Summary and Conclusions.

1. An epidemic of poliomyelitis is described, in which the basis of treatment was the stimulation of active movement, as follows: (*a*) in the acute stage by freedom from restrictions and the relief of pain by hot fomentations and baths; (*b*) in the later stages by the employment of every possible aid to activity.

2. No harmful result has been observed from these methods, which have been employed at this clinic in a lesser degree for many years.

3. In our opinion the patients in this epidemic, as compared with those in previous years, (*a*) have recovered from the reversible effects of the disease in a shorter time, (*b*) have shown a pronounced absence of tendency to deformity, vasomotor disturbances and persistence of muscular spasm, (*c*) have had a much happier stay in hospital and have maintained a high standard of morale.

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TRACHEOTOMY IN BULBAR POLIOMYELITIS.

By L. G. DEITHE,
Medical Registrar, Royal Alexandra Hospital for Children, Sydney.

BULBAR POLIOMYELITIS is becoming more frequent. During the years 1941 to 1949 at the Royal Alexandra Hospital for Children there were 43 cases of bulbar poliomyelitis out of a total of 866 cases of poliomyelitis. During the present poliomyelitis epidemic, from June 30, 1949, to December 31, 1951, there have been 46 cases of bulbar poliomyelitis out of a total of 390 proven cases of poliomyelitis. Kelleher, in England, has also noticed a similar increase both in severity and in incidence of bulbar poliomyelitis in recent years. Bower, of Los Angeles, reports 783 cases of bulbar poliomyelitis out of 4774 patients treated for poliomyelitis at his hospital during the years 1946 to 1948. During the same period, Brown, of Minneapolis, reports that slightly less than one-third of the total number of cases of poliomyelitis encountered at that centre were cases of bulbar poliomyelitis.

The more severe forms of bulbar poliomyelitis have been frequently fatal, and even with the introduction of adequate and efficient methods of artificial respiration there was still very little reduction in the overall mortality figures. In the years 1945 to 1948, at the Royal Alexandra Hospital for Children, of 12 children treated for severe bulbar poliomyelitis, 10 died and only two survived.

It was not until the introduction of tracheotomy by Galloway that any real steps were made in reducing to any degree this very high mortality rate. His original article was published in 1943 and met with a mixed reception. Since then he has further elaborated upon his methods (1947 and 1949), and in 1949 he reported a series of 15 patients suffering from bulbar poliomyelitis and treated by himself without a fatality. Bower states that since 1946 he has treated 284 patients with bulbar poliomyelitis, on 233 of whom he performed tracheotomies. He finds that the results obtained are better if the tracheotomy is carried out before anoxia develops. Mitchell and Hill, of Iowa, during 1949 performed tracheotomies on 10 patients suffering from bulbar poliomyelitis, four of whom died. Strobel and Canfield, reporting five deaths among ten patients on whom tracheotomy was performed, consider that each of the five survivors owes his or her life to the tracheotomy, and that earlier tracheotomy in three of the five fatal cases might have been life saving.

The majority of children with bulbar poliomyelitis whom we have been able to save have been left with very little residual paralysis. The prognosis in these cases is much better than in those of widespread spinal paralysis.

Rationale of Tracheotomy.

The primary difficulty in bulbar poliomyelitis is the interference with the swallowing mechanism. Owing to this, accumulation of secretion, food, fluid and vomitus occurs in and about the airway, resulting in respiratory obstruction. This respiratory obstruction affects the patient in two ways. Firstly, it causes anoxia, due to decreased respiratory tidal volume, which in turn leads to increased capillary permeability of the pulmonary vessels with a resulting tendency towards pulmonary œdema. This further increases the respiratory embarrassment and decreases the alveolar gaseous exchange. Secondly, the patient increases his respiratory efforts in the attempt to overcome the obstruction, and this tends to lower the intrathoracic pressure and increase the likelihood of acute pulmonary œdema.

The volume of salivary secretion normally is 1000 to 1500 millilitres per day, and it often increases in some diseases of the central nervous system or in nausea. This volume is also increased by any food or fluids injudiciously given to a patient with a deficient or absent swallowing reflex. A greatly diminished or absent cough reflex tends to increase the amount of stagnating fluid present. Simple

postural drainage and repeated pharyngeal suction are sufficient to maintain an adequate airway only in the milder cases. In the more severe cases, in which only effective pharyngo-laryngo-tracheal toilet can maintain an adequate airway, tracheotomy is indicated.

Tracheotomy reduces the volume of the respiratory "dead space", which in a normal person is approximately 150 millilitres. Plum and Wolff have recently shown that a patient can have a tidal volume of 200 millilitres, which is 33% to 50% of normal, and yet develop acidosis. In the example which they quote only 25% of the tidal air reaches the alveoli. If, for instance, the respiratory "dead space" is halved after tracheotomy, then 62·5% of the tidal air reaches the alveoli. So it can be easily seen that a patient with a tracheotomy can be quite comfortable with a smaller tidal volume than that of a normal person. This explains in part how some of these patients are able to carry on without a respirator after tracheotomy.

Indications for Tracheotomy.

The following are the indications for tracheotomy, as given by Galloway in 1947. (i) Fluid in the upper airway, with signs of anoxia, increasing in spite of oxygen administration, postural drainage and aspiration. Restlessness, dyspnœa, cyanosis, disorientation and mental depression are important determinants. (ii) Unconsciousness or severe restlessness in a patient not responding to other treatment in a few minutes. (iii) Pronounced restlessness or stupor in a patient in a respirator, even if he apparently has a spinal type of paralysis. (iv) Fluid accumulation not otherwise easily and certainly taken care of in a patient requiring a respirator. (v) Rapidly progressing bulbar symptoms. (vi) Bilateral spasm or paralysis of the vocal cords. (vii) Grave signs of vasomotor failure. (viii) Untrained or inefficient attendants, inadequate or poor cooperation of the patient, with the doubt that the airway will be kept constantly free of secretion.

Management of Treatment.

Tracheotomy.

Tracheotomy is best performed early and before the patient shows any considerable degree of anoxia. It should be regarded as an operation of election and not as a final resort. Also, in view of the technical difficulties of tracheotomy in a respirator, it behoves the medical attendant to carry out the tracheotomy before the patient reaches this advanced stage. The incision should be made through the second and third tracheal rings, so that the patient can be more easily nursed in the respirator.

The operation is best carried out over either a bronchoscope or a rubber endotracheal tube. In this way preliminary tracheal toilet can be carried out, oxygen can be blown directly into the trachea during the operation, and the firm tube provides a steady support onto which to cut down.

The tracheotomy tube should be left in until such time as the patient can swallow quite well, otherwise a tragic chain of events is quite likely to occur. In one of our cases the tracheotomy tube was removed prematurely, and seven days later, whilst having a meal, the child inhaled some food; it was only because of prompt bronchoscopy followed by further tracheotomy that the child did not die.

Postural Drainage and Aspiration.

After the operation the patient is nursed on a firm bed with the foot raised so that the surface of the bed makes an angle of at least 20° with the horizontal. It is necessary to have this degree of posturing to obtain adequate drainage. In the care of very young children it is sometimes possible to nurse these patients in the prone position and in this instance it is not necessary to have such a large posturing angle.

Frequent suction of both the pharynx and trachea should be carried out as often as it is necessary, in order to keep these areas dry. A bevelled, soft rubber tube should be used to aspirate the trachea. If any difficulty is encountered in aspiration of the thickened tracheal secretion, this can be softened by the instillation of a warmed 3% solution of sodium bicarbonate followed by isotonic normal saline solution through the tracheotomy tube. The trachea is then thoroughly aspirated with the rubber tube.

FIGURE I.
Method of administering oxygen directly over the orifice of the tracheotomy tube.

Oxygen.

A continuous flow of oxygen is usually required during the early phases. This should be humidified by being bubbled through water and then delivered over the tracheotomy tube orifice. In our cases we have used a funnel placed over the patient's neck or we have attached the rubber tubing directly to the inner tube by means of its

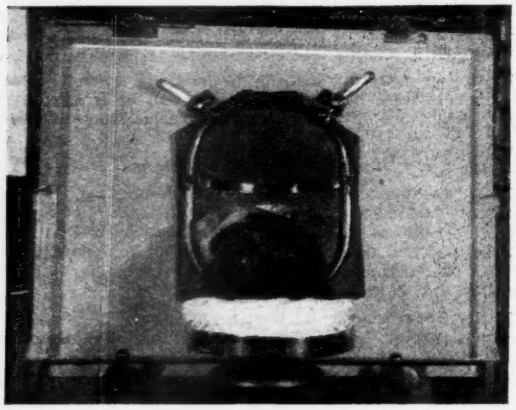

FIGURE II.
Curved steel bar used to hold rubber diaphragm away from tracheotomy tube.

lugs and delivered the oxygen through a hole cut in the rubber tubing on the side next to the tracheotomy tube orifice (see Figure I).

Maintenance of Hydration and Nutrition.

These patients are almost invariably unable to swallow and their state of hydration and nutrition has to be maintained either by intravenous therapy or by tube feeding. In considering the amount of fluid to be given it must be remembered that a considerable amount of fluid will be lost by suction of the contents of the pharynx and trachea. This amount should be carefully estimated and added to the daily requirements as calculated for each patient.

In the treatment of a normal-sized adult it would be wise to start off with about 1500 millilitres of fluid per day and gradually increase the amount up to the normal daily fluid requirements of 2000 to 2500 millilitres *plus* the amount of fluid lost by suction. In the care of children the amount of fluid to be given varies with the age and size of the child.

Considerable care should be taken not to oversalt or overhydrate the patient and thus precipitate an attack of acute pulmonary œdema, which is very likely to occur. On the other hand, dehydration will cause the tracheal secretion to become thick and viscid and difficult to aspirate.

For tube feeding a thin rubber tubing, such as a Ryle's tube, appears to be better tolerated than a thicker tube. The feedings should be given frequently and in small amounts. The patient is usually fed every four hours, a start being made with small amounts of three to four ounces gradually brought up to eight to ten ounces per feed. The patient is brought up to the horizontal position for the feeding and should be kept level for half an hour before being returned to the position of postural drainage. The tubing should be closed off between feeds to prevent aspiration of air down its lumen.

Oral feedings should not be attempted until the patient can swallow well. Care should be taken to see that the patient's food is high in protein and vitamin content. The use of concentrated protein substances and vitamin concentrates will help to simplify this problem. It is alarming to see how much weight these patients sometimes lose on what appears to be a fairly adequate diet.

Artificial Respiration.

The respirator is of great value in tiding the patient over the earlier stages. Often the respiratory musculature is overtired by the struggle against the respiratory obstruction and it needs a "breathing space" before it can capably take over its full and proper function once more. It is in such cases as these that the respirator gives such good and gratifying results.

With a "high tracheotomy" and a curved steel bar (see Figure II) to hold the sponge rubber diaphragm away from the tracheotomy tube, the tank type respirator proves satisfactory. The introduction of the portable cuirass type of respirator (see Figure III) has added another type of respirator that can be used for patients who have undergone tracheotomy. The main value of this machine is when a tracheotomy is required in a patient who is already in a respirator. The patient can be transferred to the portable machine and the tracheotomy then performed under more favourable circumstances. If necessary, the patient can be transferred back to the tank type respirator after the operation.

There are also those patients who have spinal cord involvement as well, with some degree of paralysis of the respiratory muscles. In these cases the respirator is used in the normal way as for patients with spinal cord paralysis.

In the present epidemic we have found the respirator to be of great assistance and often to have proved life saving in those cases in which the patients have been allowed to struggle for some time against severe respiratory obstruction. Frequently it has been used only to tide them over the early phase of their treatment, which often proves to be the most crucial stage of their management.

A cardinal point to remember is that artificial respiration is useless without a clear airway. Indeed, it may even be dangerous, precipitating acute pulmonary œdema and causing aspiration pneumonia.

Sedation.

Sedatives should be given once the patient has been assured of a clear and adequate airway. Not only do they allay restlessness and anguish, but they also allow the patient to settle down and become less aware of the tracheotomy tube. If the patient is in the respirator sedation will quite often help him to synchronize his respiratory movements with those of the respirator. Care must be taken not to give sedatives which have a depressant action upon the respiratory centre.

Antibiotics.

As there is usually an associated acute inflammatory process in the respiratory tree, and as possibly small areas of atelectasis are scattered throughout both lungs, it is wise to give an antibiotic to combat these processes. An antibiotic with a wide antibacterial spectrum is required, and at this hospital use has generally been made of chloramphenicol in full therapeutic doses. This can be given orally, by tube, or if necessary *per rectum*. This type of antibiotic is to be preferred to those which have to be administered by intramuscular injection in a disease in which muscle trauma may play some part in determining the extent and amount of paralysis that is going to occur.

FIGURE III.
The Monaghan portable respirator.

Results.

In the period from June 30, 1949, to December 31, 1951, 390 patients with proven acute anterior poliomyelitis have been treated at the Royal Alexandra Hospital for Children. Another ten patients have shown the clinical picture of the condition, but have not had cerebro-spinal fluid changes or developed permanent paralysis, so these patients have not been included in the series.

All told, there have been 12 deaths from all types of poliomyelitis. Of the 12 deaths that have occurred, 10 were deaths of patients suffering from bulbar symptoms.

The first tracheotomy for bulbar poliomyelitis to be performed at the Royal Alexandra Hospital for Children was successfully carried out in May, 1950. Since then a total of 13 tracheotomies have been performed, resulting in six deaths. All these patients were severely ill and had not responded to conservative treatment. This result shows a reduction in the mortality rate for patients with severe bulbar poliomyelitis treated between the years 1945 and 1948.

Reports of Cases.

The following three case reports are representative of the type of patients that have been treated by tracheotomy. The first two patients survived and the third patient succumbed very rapidly, despite tracheotomy.

CASE I.—A boy, aged seven years, was admitted to hospital with a history of having had "a cold" and a headache six days previously. These had decreased over the subsequent two days. The child was fairly well until twenty-four hours prior to his admission to hospital, when he began to vomit and again complained of headache. During the night his parents had noticed that his respirations were "rattly". On the morning of his admission to hospital his voice was indistinct, he was having difficulty in breathing and he was still vomiting. The child had not been able to pass urine since an early hour that morning. On examination he was found to have audible "gurgles" in his throat. Neck stiffness was present, his voice was nasal and weak and his palate was immobile. His intercostal muscles were weak.

He was nursed in the "head down" position and his pharynx was sucked out frequently. A Ryle's tube was inserted for his feedings and chloramphenicol therapy was begun. He appeared to settle down during the afternoon and early evening, but as night came on the pooling of secretion became much more pronounced and his respiratory movements became more laboured. An adequate airway could not be maintained despite frequent pharyngeal toilet, so tracheotomy was performed. Blood-stained purulent exudate was aspirated and the child settled down well and was soon asleep. He remained well until the following night, when the movements of his intercostal muscles became weak. The child was placed in the respirator and his general condition improved rapidly. He was taken out of the respirator for ten minutes on the following day. The time spent out of the respirator was increased by a quarter of an hour each day. He began to take his food orally after eight and a half weeks and the tracheotomy tube was removed two weeks later.

He was given speech therapy and physiotherapy, and he made a steady gradual recovery. Now, except for a nasal voice and weakness of the flexors and extensors of the left side of his neck, he is well (see Figure IV).

CASE II.—A boy, aged seven years, came home three days prior to his admission to hospital complaining of headache and sore throat. He was put to bed, and two days later his mother noticed that his voice was nasal and weak. This condition became worse during the day, and that afternoon he was unable to swallow and fluids were regurgitated through his nose. Another child in the same class at school as the patient had died of poliomyelitis eight days earlier. Physical examination of the patient revealed a nasal voice, neck rigidity, left palatal paresis and weakness of the right side of the face. Lumbar puncture produced faintly opalescent fluid containing 15 leucocytes per cubic millimetre; 60% of these cells were mononuclear cells. The protein content was 20 milligrammes per 100 millilitres, the chloride content was 760 milligrammes per 100 millilitres, and Benedict's test gave a positive result for glucose. There was no growth of micro-organisms on attempted culture of the fluid.

He was given tube feedings and the foot of the bed was elevated. He required only a small amount of suction for the first three days, but it was then noticed that the amount of pooling was increasing and hourly suction was required. He was twice placed in the portable respirator with little improvement, so tracheotomy was then performed. Thick mucus was aspirated from the trachea. He was placed in a tank-type respirator and immediately settled down. His colour improved rapidly and his pulse rate quickly fell. The following day the tracheal exudate was much thinner and he was able to spend a short time out of the respirator. Within two weeks he was spending only a couple of hours a day in the respirator. One month later he was taking food by mouth, and shortly after this his tracheotomy tube was removed.

Physiotherapy and speech therapy were started about this time, and now the child has only a mildly nasal voice and weakness of the extensors of his right great toe and of the peronei of his right leg (see Figure IV).

CASE III.—A boy, aged nine years, two days before his admission to hospital, complained of anorexia. The following morning he was noticed to have a stiff neck, and that night he began to regurgitate fluids through his nose. He began to have difficulty in swallowing, and he vomited everything that he took by mouth. His voice was also noticed to be nasal. Whilst being brought to the hospital by ambulance he began to find it difficult to breathe and commenced to shiver. Examination showed a large amount of thick mucus in his pharynx, and he was cyanosed, with deep labouring respirations. He had a stiff neck and a paralysed soft palate.

Immediate tracheotomy was performed and blood-stained mucus was sucked out of the trachea. His colour improved with the clearing of his airway and he was put straight into the respirator. The original tracheotomy tube was too small, and the patient was taken out of the respirator whilst a larger tube was being inserted. During this procedure he suddenly collapsed, brownish fluid welled up through the tracheotomy tube and the patient rapidly became very cyanosed and died despite sucking out of the trachea and his being put back in the respirator.

Post-mortem examination revealed histological changes consistent with acute anterior poliomyelitis throughout the spinal cord and *medulla oblongata*. The changes were least in the lumbar cord and became more pronounced as the cord was ascended, the maximal changes being found in the medulla. There were no changes to be seen in the cerebral cortex. Examination of the lungs revealed generalized œdema and small foci of bronchopneumonia.

Summary.

Bulbar poliomyelitis is becoming a more frequent disease. The severe forms of the condition carry a high mortality rate. Survivors often show little disability.

Early tracheotomy is the treatment of choice for those patients who develop respiratory obstruction. Often this is used in conjunction with the respirator.

FIGURE IV.

The two patients (Case I, left; Case II, right). This photograph was taken approximately five months after the onset of their illness.

The histories of three patients are reported. Two recovered after tracheotomy, whilst the third died after the procedure.

Acknowledgements.

I wish to thank Dr. R. L. Stephen for permission to publish the histories of the first two patients, and Dr. W. P. MacCallum for permission to publish the history of the third patient, and also the chief resident medical officer, fellow registrars and resident medical officers, who at various times helped with these patients. Last, but not least, I should like to stress the important part played by the sisters and nurses of the Royal Alexandra Hospital for Children in the successful treatment of these patients.

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POLIOMYELITIS AND ATMOSPHERIC CONDITIONS IN CERTAIN AUSTRALIAN CITIES.

By D. J. R. SNOW,
Perth.

THAT outbreaks of acute anterior poliomyelitis appear to have some relation to season is an old observation. It has long been recognized that it is predominantly a disease of warm weather. Thus the majority of European outbreaks have developed in the summer and autumn; in America, summer has been, in the main, the epidemic season; and, in Australia, the warmest months have usually shown the highest incidence of cases.

In general, it has been noted that epidemics tend to subside with the advent of cold weather; but there have been a few examples of epidemics persisting into and throughout the winter. Epidemics actually beginning in the winter have been extremely rare.

Occasionally it has been reported that the season either preceding or accompanying some epidemic has been unusual. Thus, over forty years ago, Litchfield (1904) observed that a small summer outbreak in Sydney accompanied a particularly wet and cool summer; while Stephens (1908) commented as follows upon an epidemic of 135 cases in Victoria: "The outbreak supervened after an excessively dry warm summer, with a dearth of water in the catchment area such as had not been experienced for many years." This epidemic occurred during the months of April, May and June. Dick (1925), in reviewing several aspects of the disease in Australia, said: ". . . warm weather if not a predisposing cause of the disease, is its usual concomitant. . . . It should be noted, however, that outbreaks of epidemic proportions have occurred in cold countries like Norway and Sweden in the winter season."

Helms (1941), in a description of an epidemic involving 711 cases in New South Wales from October, 1937, to September, 1938, indicated a relationship between temperature and absolute humidity on the one hand, and increase and decrease of cases of poliomyelitis on the other.

Bull (1939), in the report on the Melbourne epidemic of 1937, described unusual weather in the form of lower temperatures, less radiation and more fogs.

Finally, during the Western Australian epidemic of 1948 (Commissioner of Public Health for Western Australia, 1948), involving 311 cases over a period of approximately nine months, the monthly incidence of poliomyelitis was very closely related to the mean monthly temperature and humidity (the latter being expressed as "saturation deficiency").

No other published references to an association between weather factors and poliomyelitis epidemics in Australia appear to be available.

In other parts of the world, particularly in America and Europe, the apparent influence of the weather on epidemic poliomyelitis has similarly excited comment, and the results of several studies have been published. These have been conflicting and inconclusive. However, the possibility of a relationship has never been frankly discounted, and at least two eminent authorities have recommended studies into this aspect of poliomyelitis.

Sir Macfarlane Burnet (1940) remarked: ". . . it seems likely that close investigation of the influence of climate and other environmental factors on the course of epidemics might suggest practical measures of individual protection." Rhodes (1947), in the course of his most comprehensive review, states: "Further information on the possible effect of meteorological conditions on the incidence of poliomyelitis would be of interest. For example, the study of saturation deficiency might throw some light on the importance of droplet infection."

Accordingly, during the Western Australian epidemic of 1948, meteorological conditions were carefully studied, saturation deficiency being given special attention. After daily readings, weekly records and monthly means had been examined, it was ascertained that the last-mentioned provided the most useful data for comparison, and they were therefore adopted as a basis for investigation. The resultant findings have been fully described in the Annual Report of the Commissioner of Public Health in Western Australia for 1948. A close association was found between the incidence of poliomyelitis on the one hand, and saturation deficiency, mean temperature and barometric pressure on the other. The graphs which accompanied the report showed clearly that the epidemic waxed with increasing humidity and diminishing temperature, and waned as drier and warmer conditions returned.

The seasonal behaviour of this particular epidemic was peculiar, in that the epidemic gathered momentum as winter advanced, and indeed reached its peak in midwinter. Epidemics of poliomyelitis in other parts of the Commonwealth and in other parts of the world have seldom behaved in a similar way. The epidemic, therefore, while not being unique, was at any rate unusual.

Unfortunately at that time meteorological records for other capital cities during epidemic periods were not available for comparison, and it was therefore not possible to assess the value or otherwise of the Perth observation. Nevertheless the mathematical correspondence between the epidemic and the weather was so striking that further inquiry along similar lines was planned in the event of subsequent epidemics elsewhere.

Since then Perth has been spared a major outbreak, but three very large epidemics have overtaken the cities of Adelaide, Melbourne and Sydney respectively. The incidence of the disease at these centres has been secured from the various State health departments concerned, while the meteorological data for all these cities have been obtained from the Sydney Weather Bureau. The incidence of poliomyelitis by months, the saturation deficiency and the mean monthly temperature are shown in Table I, and have also been plotted as graphs.

It will be seen that in each of these three epidemics there appears to be an association between the disease and the weather, but it will be noted that the association is very different from that found in Perth. In fact, the epidemic waves correspond with weather changes which are precisely the reverse of those observed in Perth. Rising incidence accompanies falling humidity and rising temperature, and as moist, cool conditions advance, the epidemic subsides. This, of course, is said to be the usual experience with epidemics of poliomyelitis. The feature is particularly pronounced in the case of Adelaide, is less pronounced in Sydney, and is considerably modified in Melbourne, where the respective peaks are separated by an interval of four months, but the general trend in all three instances is similar.

Comparable studies have recently been undertaken in America, and Armstrong (1950 and 1951) has published the results of his investigation into the relationship between atmospheric conditions and poliomyelitis in several localities in the United States. He, too, has found a close association between temperature and humidity on the one hand and the incidence of poliomyelitis on the other, and has suggested that a relative humidity of 27% to 28% for atmospheric air warmed to 90° F. may represent a critical level below which poliomyelitis does not readily spread. He has further suggested that a study of atmospheric conditions may make it possible to predict the course of an established outbreak of poliomyelitis for about three weeks in advance.

Discussion.

Before we proceed to a discussion of the possible mechanisms whereby atmospheric conditions may influence epidemic poliomyelitis, it may be well to clarify the term "humidity".

"Relative humidity", which is perhaps the commonest way of expressing the moisture content of the atmosphere, may be defined as the ratio of the actual amount of water in a given volume of air to that which would be present were the air saturated, and it is expressed as a percentage.

"Absolute humidity" is the mass of aqueous vapour, expressed in grammes per cubic metre of air.

"Saturation deficit" is the difference between the actual vapour pressure and saturation vapour pressure at any given temperature. It is said to be a more satisfactory measure than relative humidity.

In general the relative humidity of cool air is greater than the relative humidity of warm air; but the latter has in fact a small evaporating power so far as the upper respiratory passages are concerned. When the cool air of winter is breathed, it is warmed by the respiratory tract and its volume increases so that its relative moisture content becomes smaller. In any event cool air can hold less moisture than warm air. Hence it is in fact drier than the warm air of summer. Winter air thus tends to dry off the secretions in the naso-pharynx, concentrate them, and stimulate the production of fresh secretion (which, of course, is the common subjective experience in winter). Summer air on the other hand more nearly approaches the temperature and moisture content of expired air (90° F., and 90% relative humidity), and therefore has an insignificant effect on these secretions.

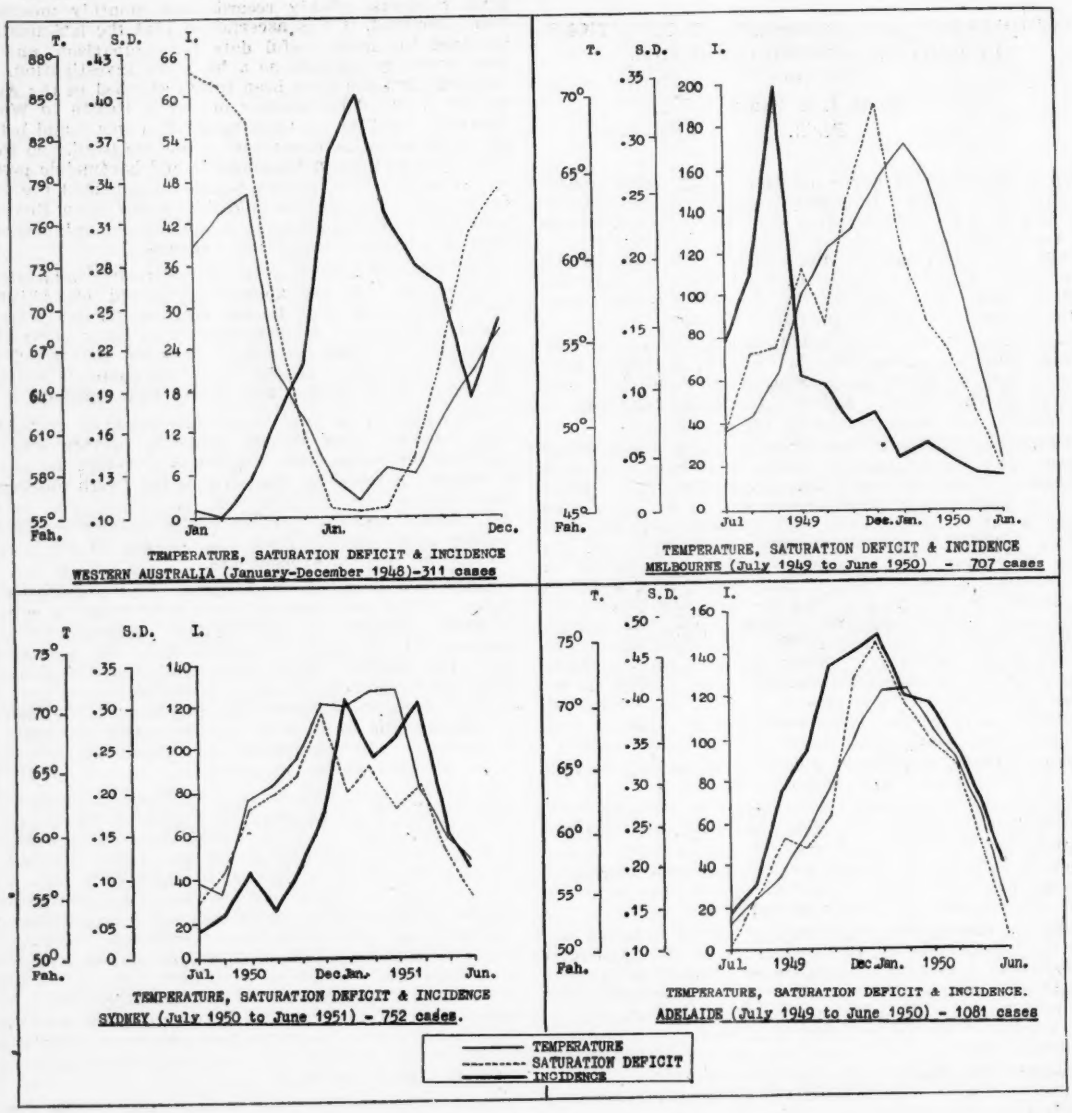

FIGURE I.

"Relative humidity", which is perhaps the commonest way of expressing the moisture content of the atmosphere, may be defined as the ratio of the actual amount of water

It is possible that atmospheric conditions can influence the prevalence of poliomyelitis in at least four ways: (i) Temperature and humidity may influence the viability of poliomyelitis virus in a given environment. (ii) The

TABLE I.

Incidence of Poliomyelitis, Mean Monthly Saturation Deficit, and Mean Monthly Temperatures for Twelve Consecutive Epidemic Months in Western Australia, Sydney, Melbourne and Adelaide respectively.

Western Australia.			Sydney.			Melbourne.			Adelaide.		
Number of Cases.	S.D.[1]	T.[1]	Number of Cases.	S.D.	T.	Number of Cases.	S.D.	T.	Number of Cases.	S.D.	T.
1	0·416	74·1	15	0·065	56·1	80	0·059	49·8	19	0·102	52·4
0	0·407	76·5	23	0·105	55·1	113	0·199	50·6	32	0·165	54·5
5	0·381	76·9	40	0·177	62·4	119	0·126	53·1	75	0·230	56·7
14	0·222	65·4	25	0·198	63·7	62	0·184	57·9	94	0·215	60·1
22	0·158	62·0	42	0·218	65·5	58	0·162	60·5	132	0·256	63·5
52	0·108	57·8	69	0·293	69·7	40	0·246	61·7	140	0·416	67·9
60	0·105	56·3	122	0·198	69·9	46	0·316	64·9	148	0·468	70·0
43	0·107	58·4	95	0·226	71·4	24	0·204	66·5	119	0·382	70·4
36	0·142	58·0	105	0·176	71·8	29	0·146	64·4	116	0·339	67·5
33	0·217	62·1	120	0·205	63·2	25	0·127	59·3	96	0·310	65·0
17	0·303	65·1	58	0·126	59·7	16	0·082	53·7	71	0·205	60·3
28	0·333	68·2	38	0·067	57·0	15	0·030	48·0	39	0·113	53·8

[1] "S.D." = saturation deficit; "T" = temperature (degrees Fahrenheit).

persistence of infective droplet nuclei as air-borne masses may be influenced by atmospheric humidity and air movement. (iii) The general susceptibility or resistance of an individual may fluctuate with atmospheric variations. (iv) The physiological condition of the upper respiratory mucous membrane may be influenced by atmospheric air inhaled. The last of these factors is of particular interest. The upper part of the respiratory tract is believed to be one of the portals of entry of poliomyelitis virus. The virus is known to be present in oro-pharyngeal washings in the early stages of the disease and can presumably spread by droplet. Mucous secretion is said to have a protective action against microorganisms.

Armstrong has developed an attractive hypothesis to explain the preponderantly summer prevalence of poliomyelitis. The essential features are as follows: (i) Atmospheric conditions influence both quality and quantity of naso-pharyngeal secretion through a drying effect. (ii) Humidity is more important than temperature in determining the degree of this drying effect. (iii) Inspired air is adjusted to a temperature of 90° F. and a relative humidity of 90% within the respiratory passages. (iv) The cool air of winter is relatively less humid than the warm air of summer (adjusted to 90° F.). (v) Winter air therefore tends to dry up and concentrate surface secretions and encourage additional secretion. Summer air causes minimal changes in these secretions, which thus do not hinder the virus unduly.

This concept is applicable to summer epidemics of poliomyelitis in Australia, but does not explain the Perth epidemic or other exceptional epidemics which have occurred in winter. It must be remembered, however, that atmospheric conditions and the portal of entry represent only one facet of a very complex problem. The previous experience of the community, the population density and the strain of virus are but a few of several factors which could be advanced to explain exceptional outbreaks which fail to conform to the usual summer pattern.

Conclusion.

There has been an apparent relationship between atmospheric conditions and poliomyelitis epidemics in four Australian capital cities during the last four years. This relationship has not been of a consistent type, but further studies are warranted. The suggestion that atmospheric conditions may influence poliomyelitis prevalence through the portal of entry of the virus is worthy of more attention.

Acknowledgement.

I am indebted to Mr. Colin Waldon for preparing the graph.

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RESPIRATORY INSUFFICIENCY IN POLIOMYELITIS.[1]

By S. E. J. ROBERTSON, M.B. (Sydney), M.R.C.P. (London), M.R.A.C.P.,

Honorary Assistant Physician, Royal Alexandra Hospital for Children, Sydney.

BEFORE respiratory insufficiency in poliomyelitis can be discussed, it is important to define the various types which may be encountered, either singly or as mixed forms. The first and commonest type is that in which there is weakness or paralysis of the respiratory muscles, the intercostals or the diaphragm, and which follows damage to anterior horn cells in the cervical and thoracic segments of the spinal cord. The second type is that in which paralysis of the muscles of the pharynx occurs and is a common manifestation of virus lesions in the motor nuclei of the bulb. If this type is present without damage to the anterior horn cells subserving the respiratory muscles or to the respiratory centre, interference with normal breathing is due solely to the accumulation of secretions in the pharynx, which obstruct air entry and which the patient is unable to swallow. The third type, very rarely encountered, is bilateral abductor palsy of the vocal cords from involvement of the vagus nuclei subserving this function. Finally, there is damage to the respiratory centre or centres in the

[1] Based on observations made during the tenure of a travelling fellowship of the Post-Graduate Committee in Medicine in the University of Sydney, and read at a meeting held by the Institute of Child Health of the University of Sydney at the Royal Alexandra Hospital for Children on March 21, 1952.

medulla oblongata leading to loss of rhythmic control of breathing. This last-mentioned has been said to be always secondary to anoxia following pharyngeal or respiratory muscle paralysis, but the severe specific lesions found in this portion of the brain stem in fatal cases of this type would seem to be against this.

Differential diagnosis may be extremely difficult and almost impossible when mixed types are encountered. Weakness of the intercostal muscles or diaphragm should always be looked for most carefully, but such signs as cyanosis, mental confusion, twitching of the *alæ nasi* and use of the accessory muscles of respiration do not appear till many neurons are out of action and the remainder are being grossly overstimulated by oxygen lack and carbon dioxide retention. This overuse of the remaining active respiratory muscles is very likely to lead to further damage to the anterior horn cells of the motor nerves supplying them. Therefore it would seem desirable to endeavour to make an early diagnosis of weakness of these muscles, so that the patient can be placed in a tank type of respirator as soon as possible. Efforts have been made to do this by estimating the vital capacity, and a rough but effective method is to see how long the patient can count after a full inspiration. Another way is to ask the patient to inflate the bag of an anæsthetic machine and to estimate the increase in its volume by water displacement. The most accurate method is to use a freely moving spirometer, but studies of vital capacity in normal children of various sizes are needed. Even if the normal vital capacity of the patient is not known, serial estimations, which are diminishing in volume, would be the signal for placing him in a tank respirator. I can see obvious difficulties in estimating the vital capacity in young children, but the procedure should be applicable to older subjects.

The distinction between pharyngeal paralysis and disturbance of the respiratory centre is almost impossible in most cases. Both conditions will cause irregular ineffective breathing due, in pharyngeal paralysis, to accumulated secretions intermittently obstructing inspiration and, in central depression, to loss of rhythmic stimulation of breathing. If respirations are irregular and secretions are not collecting in the pharynx, a central disturbance can be diagnosed. When breathing becomes extremely slow or hiccupping in type, damage to the respiratory centre becomes obvious.

Bilateral abductor paralysis of the vocal cords in the larynx will result in acute inspiratory obstruction. This can be confirmed by direct vision with a laryngoscope.

Of recent years there have been a great many advances in the treatment of respiratory insufficiency in poliomyelitis. These advances have applied to methods of ensuring efficient respiration and to apparatus used to bring about the desired result.

One of the difficulties yet to be overcome completely is that of the long-term respirator patient who has become addicted to greater alveolar ventilation and lower alveolar carbon dioxide tension than normal. The origin of this may be physiological, because the respiratory centre has become accustomed to function at a lower plasma carbon dioxide level. On the other hand, it may be psychological and be part of a fear reaction. However, it is unlikely to develop if the machine is run at the minimal level sufficient to produce proper alveolar ventilation. Verbal reports of the subjective sensations of the patient are unreliable, and the best giude is to watch the patient's face and nostrils and his own respiratory efforts during sleep while the power of the machine is gradually reduced, the rate remaining the same.

The dangers of alkalosis from over-ventilation have probably been overemphasized, as moderate degrees can be compensated for by the kidneys secreting a more alkaline urine. However, greater degrees will produce anxiety, confusion and a sense of suffocation, which will lead to greater overbreathing. Tetany has been produced on occasions, and a reduction of arterial carbon dioxide content is believed to cause cerebral vasoconstriction and increased delta waves in the electroencephalogram. In some subjects, who have been rendered alkalotic, spasticity of the muscles of the shoulders and chest has been noticed, and this may be responsible for the "rigid" chests which occur in some respirator patients.

The rate of respiration in the respirator should be that which is normal for that particular patient, unless fever or respiratory obstruction is present, which necessitates a faster rate. The Both respirator has the weakness of not being able to work at rates of less than 20 per minute, and this may be a definite disadvantage with larger children, in whom a rate slower than this may be necessary to prevent overventilation. This question of hyperventilation will never be satisfactorily settled until there are some means of making instantaneous readings of the alveolar carbon dioxide tension.

While hyperventilation is to be avoided, underventilation with resultant oxygen lack is even more dangerous. Oximeter readings and oxygen saturation levels can indicate only rather severe levels of oxygen deficiency. Therefore it would seem best to choose a minute volume for the patient that is rather greater than his theoretical requirements at rest. Extra oxygen should be provided by means of a tent, mask or catheter when oxygen lack exists. It should be used routinely with any respiratory difficulty or bulbar involvement, and when there is any evidence of encephalitis which may be due to, or be aggravated by, oxygen lack. If a tent or mask is used it must not be allowed to prevent close observation of the patient or easy approach to the throat for aspiration. Nasal instillation of oxygen is the simplest, and oxygen supplied in this way will be less likely to irritate or cause caking or stringiness of secretions if it has first been passed through warm water. Finally, it must be emphasized that oxygen therapy will not prevent carbon dioxide retention with its attendant dangers.

A room respirator has been developed which allows attendants to enter by a door and which can accommodate several patients. Complete physical examinations can be carried out on patients in this apparatus, samples of blood taken and intravenous infusions given easily. It is economical in operation, but it is costly to erect and occupies much precious space which will be used only during epidemics.

The cuirass type of respirator is now standard equipment in most hospitals, fitting either all around the thorax and abdomen or only in front. It has only a minor action on the intercostal muscles, acting most efficiently on the diaphragm, which, of course, must not be in a state of spasm. It offers great psychological advantages in cases of chronic respiratory paralysis and in weaning patients away from a tank respirator. However, it is quite unreliable in the acute stage of respiratory paralysis, which may quickly progress, but it can be life-saving in transport of patients to a tank respirator.

Similar advantages and disadvantages have been found in the use of the rocking bed, which acts by causing the abdominal contents alternately to push against and pull away from the diaphragm. It is not good enough for severely paralysed patients or for those in the acute progressive stage, but it can be used in mild cases provided the diaphragm is not in spasm and there is not much abdominal distension.

Positive pressure over the mouth and nose applied synchronously with negative pressure of the tank respirator is being extensively used. While it is in action aspiration of the throat is impossible, but it is effective if used alone for short periods while the tank respirator is opened for essential procedures. An Oxford inflator or a McKesson resuscitator is convenient. The application of a face mask for long periods is often not tolerable to the patient, and besides, the lips cannot be inspected for cyanosis. These last two problems have been solved by the attachment of transparent airtight domes to the head ends of tank respirators. Positive pressure can then be applied within the dome, either synchronously with the negative phase of the tank respirator's action or to maintain the respiratory exchange of the patient while the tank is opened.

Great advantages have been claimed for positive pressure respiration in reducing or preventing acute pulmonary

œdema, which is particularly likely to occur in patients in a tank type respirator when partial obstruction greatly increases the intrathoracic negative pressure. However, from the point of view of pure physics, there can be no difference between positive pressure applied over the nose and mouth and negative pressure applied over the thorax, if no obstruction to inspiration is present. Positive pressure respiration is also said to promote bronchial drainage, produce more uniform alveolar dilatation, protect against atelectasis and increase lymph flow from the lungs. The fact that a tank type respirator applies rhythmic negative pressure to the abdomen and limbs may have some bearing on increased circulation, but no increased venous return to the right side of the heart has been shown while it is in action.

When a patient in a respirator is unable to cough owing to diaphragmatic paralysis, inability to take a deep breath or paralysis of the glottis, the removal of secretions from the trachea and bronchi becomes a problem. Devices are being developed to enable a patient to cough effectively under these circumstances, but until these reach the stage of clinical use the provision of positive pressure within the tank respirator at some stage of the respiratory cycle often makes coughing easier.

The most effective way to deal with increased secretions in the trachea and bronchi is by posture, and particularly changes of posture. The supine position has been generally used because it is more comfortable for the patient and nursing is easier. Should secretions have to be aspirated, however, the bottom of the respirator has to be elevated to make an angle of at least 20° with the horizontal, and an angle of 35° is used as a routine in some clinics. Placing the patient at these angles is likely to cause puffy eyelids and papillœdema, and very probably cerebral œdema occurs. To avoid this the return to the horizontal position for a short period in each hour is advised. It is important to realize that, if the patient is lying prone on a horizontal surface with the face tilted to one side and the chin one and a half inches below the level of the chest, there is a slope of 15° from the carina of the trachea to the corner of the mouth. Therefore, in this position very little tilting of the respirator is necessary to attain satisfactory drainage of the trachea and pharynx. To drain the various bronchi is more difficult and can be adequately effected only by frequent changes of posture. The patient can be placed in one or other lateral position for a period of two hours without much discomfort. No change in the tidal air has been found when the patient is shifted from supine to prone or lateral position. If the prone position is used, combined with frequent changes of position, segmental collapse should not be the frequent complication of respiratory insufficiency that it is at present. Similarly, if great degrees of tilt are avoided, upper lobe collapse is unlikely to occur. These changes of position are made a lot easier by the iris type of collar, which is easily opened while the patient is moved, and is much more comfortable. A simple fitting is necessary to accommodate a tracheotomy tube if this operation should prove necessary. Before passing on to the subject of pharyngeal paralysis, I shall briefly mention the subject of hot packs. They are used mainly for pain and tightness of peripheral muscles, but there are occasional times when respiratory insufficiency is due to spasm of the intercostal muscles, and hot packs will cause freer movement of the chest wall.

Two important principles are fundamental in the treatment of pharyngeal paralysis. The first is the prevention of accumulation of pharyngeal secretions, which make regular respirations impossible. It is this irregular respiratory action in pharyngeal paralysis which makes its differentiation from depression of the respiratory centre so difficult. The second is the provision of adequate rest for the patient, as fatigue, if excessive, will almost certainly lead to a fatal outcome.

Unless there is coexistent paralysis of the respiratory muscles patients with pharyngeal weakness should not be placed in a respirator. They should be laid in the prone position on a bed with the neck slightly flexed and the chin to one side. The foot of the bed should be elevated to aid drainage from the trachea, but not to any great degree, so that a rise in intracranial pressure will be avoided. This posture will result in adequate drainage if the secretions are thin, but in severe cases will have to be aided by pharyngeal suction. Aspiration by suction must never be carried out roughly, as any clumsiness will injure the mucous membranes, frighten the patient and increase the amount of secretion. With children one must be firm but particularly gentle, and the procedure is most effective with the phlegmatic type of child. A metal aspirator is more effective than a flexible rubber one, as the tip of the former can be more accurately placed. The tip should be placed on the base of the tongue just in front of the larynx, but not touching the posterior pharyngeal wall to avoid stimulating the gag reflex. Small amounts of saline or sodium bicarbonate should be instilled during suction into the pharynx or, if tracheotomy has been performed, into the trachea, to make the mucus less tenacious.

To carry out the second principle of treatment substantial sleep for long periods is necessary, and postural drainage and aspiration may not be effective in producing this. In this case tracheotomy may be dramatically effective. Tracheotomy has been advocated as a routine procedure in pharyngeal paralysis, to prevent a severe choking attack with its attendant acute anoxia, which will be added to the effect of the virus on the central nervous system. However, good nursing will often make this operation unnecessary. In the rare event of bilateral abductor paralysis, tracheotomy is immediately indicated. Tracheotomy has been objected to on the grounds that it is an ordeal for the patient, who needs rest above all, that the mortality after it in poliomyelitis is very high, and that it increases pharyngeal secretions. It is certainly not indicated when there is merely inability to cough or swallow, when there is laryngeal involvement except bilateral abductor paralysis, or when there is pulmonary œdema or atelectasis. Special attachments for the tracheotomy tube are available which make the maintenance of adequate oxygen tension easier and provide a means of securing positive intrapulmonary pressure in the case of œdema. The provision of oxygen in adequate amounts without such an attachment is impossible unless a suitable dome is available.

During the acute febrile stage even tube feeding should be avoided owing to the enhanced tendency to vomit. With complete pharyngeal paralysis parenteral administration of fluids can be used for five to seven days. Gavage can be started as soon as the danger of vomiting is past. In pharyngeal paralysis there is a great tendency to recovery; functional recovery, sufficient to prevent aspiration, is attained in a week or so in most cases, and ordinary feeding can be cautiously resumed in a few weeks.

Central disturbances due to involvement of the respiratory centre by the virus are extremely difficult to treat successfully. The patient is usually semiconscious and breathes with shallow irregular movements, but can, as a rule, take a deep breath on demand. Involvement of the adjoining vasomotor or vagus centre is responsible for various changes in cardiac rhythm, which also, very probably, add to the respiratory distress by causing acute pulmonary œdema. If this last-mentioned complication occurs, the underlying condition may be indistinguishable from acute pulmonary œdema due to sudden cardiac failure, or to increase in negative pressure because of partial respiratory obstruction from pharyngeal secretions. If respiratory insufficiency is thought to be due to central disturbance alone, the respirator should not be used, as the patient's own irregular efforts at breathing prevent effective action by the machine. Various methods have been tried to counter this irregularity of respiratory stimuli. Curare may have some use in paralysing the muscles making these irregular efforts, so that a respirator will be effective. Administration of caffeine and sodium benzoate in doses of 0·25 gramme by intramuscular injection every four hours may cause more effective breathing and is worth trying. In some desperate cases morphine has been used to cause further depression of the respiratory centre, enabling successful use of the respirator, and this has been lifesaving on occasions. More usually sedatives are contra-

indicated in such acute cases, as the patient may have to make conscious efforts to breathe at any time. Electrophrenic respiration is the most promising means of bringing about regular respirations in these cases. In this the machine, by means of a manually applied or clamped electrode, intermittently applies a direct electrical stimulus to the exposed phrenic nerve or to the overlying skin. This results in rhythmic contraction of one half of the diaphragm, and there also seems to be inhibition of the patient's own respiratory efforts, if these are irregular or ineffective. The making of skin contact with the phrenic nerve is very difficult, even for the most experienced, and surgical exposure is justified in some cases. Just how long the nerve will stand this treatment is not known, nor has it been ascertained how soon the phrenic nerve will degenerate after destruction of its anterior horn cells.

It has been supposed that disturbances of cortical and medullary centres are largely due to œdema. To counter this, dehydrating solutions have been given intravenously as a routine, usually in the form of 10% glucose solution by slow intravenous drip or plasma concentrated two and a half times. These procedures are claimed to cause dehydration and to reduce the quantity of pharyngeal secretions, the aim being to produce a urine of specific gravity between 1·025 and 1·030. It is difficult to understand why such methods should not be just as effective with spinal involvement as with cerebral, and, without elaborate studies on blood volume, their beneficial effects may be due to other reasons, such as relief of peripheral circulatory failure.

In conclusion to this short review of a controversial subject, I should like to emphasize that the most important aim is to provide good nursing and to avoid any unnecessary strain on the patient. One must strenuously resist the temptation to provide some form of active treatment, whatever it is, in this terrifying disease. The entire aim of all treatment is to attain maximum comfort of the patient. Any treatment which is not indicated is quite definitely contraindicated.

Reports of Cases.

A CASE OF POLIOMYELITIS WITH FACIAL PARALYSIS FOLLOWING A DENTAL PROCEDURE.

By D. J. R. SNOW,
Perth.

ON September 13, 1951, a young departmental dental officer commenced the routine examination of children's teeth at a suburban State school in the Perth area. On the following day he inspected the mouth of a boy, aged eight years, who was one of a number of other children examined. Having obtained the consent of the various parents, the dentist proceeded with the necessary operative work, beginning on September 19, when he extracted teeth from five children. On September 20 he attended to another five, and on September 21 eight children received treatment. Among these last was the boy, aged eight years, who is the subject of this report.

Clinical Record.

At the time of the initial examination the dentist noted that the boy was of a slow, dull and lethargic type, and the teacher confirmed that the boy's disposition in class was similar. After inspection of the boy's mouth, the dentist recorded that no teeth were missing, but that the two deciduous molars in the lower jaw on the left side and an upper right deciduous molar required extraction because of advanced caries. However, no sepsis was noted, and no abnormality was observed in the throat.

On September 21, using a syringe with a 25-gauge needle, the dentist injected approximately two millilitres of "Monocaine" (which contains 1·5% of monocaine hydrochloride with one part in 100,000 of adrenaline) into the left side of the lower jaw, using the "mandibular block" technique. The precise site of the injection and the procedure as described to me were as follows:

> The needle was inserted at a point medial to the internal oblique ridge of the ascending ramus of the mandible about one centimetre above the occlusive surface of the molar teeth. It was then passed through the fibres of the buccinator muscle near the pterygo-mandibular ligament, and along the internal aspect of the ascending ramus for about half of its width and so to the point where the inferior dental nerve enters the mandibular canal at the mandibular foramen. Here the bulk of the solution was deposited, the lingual nerve being anæsthetized with about 0·5 millilitre while passing the internal oblique line, and the buccal mucosa with a similar quantity in the muco-buccal fold. Except for some resistance while penetrating the fibres of the buccinator, the passage of the needle was through soft tissue.

The two lower left molars were then extracted with ease and speed, the operation occupying no more than a few moments.

On October 5 the boy was brought to the dentist by his mother. She said that he had not been well since the extractions. He had been unduly drowsy, had slept a great deal, had had an intermittent fever, and had vomited. When questioned, the boy complained of a pain at the angle of the jaw, and on examination it was found that he was very tender in that region and was unable to open his mouth except with great difficulty, the teeth of the upper and lower jaws being separated by a distance of less than half an inch. The wounds had healed, and there was no evidence of infection. Being unable to account for the trismus or for the other signs of illness, the dentist suggested that the child be taken to a doctor. This advice was apparently not heeded until three days later, when the child was presented to a general medical practitioner. The latter elicited a history of neck stiffness, observed left-sided facial paralysis, and promptly referred him to the Princess Margaret Hospital for Children with a provisional diagnosis of Bell's palsy but a tentative reference to the possibility of poliomyelitis.

A history of fever, drowsiness and neck stiffness of about one week's duration was obtained, the existence of left facial paralysis was confirmed, and a lumbar puncture was performed. The latter revealed a clear fluid under a pressure of 210 millimetres; the protein content was 10 milligrammes per 100 millilitres and the cell content was 13 lymphocytes per cubic millimetre. The patient was then transferred to the Infectious Diseases Hospital, Subiaco, with the diagnosis of poliomyelitis.

On arrival he was medically examined for a third time. Apart from measles two years previously, the patient had not suffered from any of the other diseases of childhood. He had been immunized against diphtheria with toxoid during infancy. He had two sisters, one aged eleven years, who attended the same school, the other aged sixteen years, who worked as a shop assistant in a large department store in the city. Both were and had been well. The history of his illness according to his mother was that he had been ill ever since his dental extractions seventeen days previously. The illness had begun with drowsiness and fever; seven days later he had complained of pain in the neck, three days after this the mother noticed that his face seemed weaker on one side; finally he had vomited twice, and shortly before his admission to hospital he had complained of soreness at the back of his legs and was found to be a little unsteady on his feet.

On examination of the patient, his temperature was found to be 99·2° F., the pulse rate was 120 per minute, and the respirations numbered 24 per minute. No clinical abnormality was detected in his respiratory, alimentary or cardio-vascular systems. Examination of his neuromuscular system revealed facial paralysis of the lower motor neuron type on the left side, moderate weakness of the hamstring group of muscles on both sides, and slight weakness of abduction in the left lower limb. All deep reflexes were present and sensation was normal. There

was some pain on flexion of the head, but no frank neck rigidity.

After four weeks of bed rest, symptomatic treatment and routine physiotherapy the hamstrings had recovered much of their lost function and the child was able to walk fairly well. However, the facial weakness was still evident. The child was then returned to the Princess Margaret Hospital for Children for surveillance and further physiotherapy.

When the child was examined on January 9, 1952, facial asymmetry could still be detected at a glance. However, the muscles had evidently recovered much of their lost power. The boy was able to close his left eye completely, although the orbicularis was demonstrably weaker than its fellow on the opposite side. The left *corrugator frontalis* and the frontal belly of the occipito-frontalis were functioning fairly well, although both vertical and horizontal forehead furrows were asymmetrical. The masseter, pterygoids and temporal muscles on the affected side were strong, and the buccinator seemed normal.

All eye movements were normal and there was no abnormality of taste or hearing.

All muscle groups in the lower limbs reacted equally and fully to the usual clinical tests.

Associated Cases.

At about the time when this incident occurred the incidence of poliomyelitis in Perth seemed to have been geared to a higher level than usual. A greater number of cases were being notified every week than had been the experience for several months, and there was some apprehension that a major epidemic was about to develop.

On October 5, a boy, aged four years, was admitted to hospital with a febrile illness of a fortnight's duration and extensive paralysis of the right upper limb, chiefly involving the deltoid and supraspinati. He lived little more than a hundred yards from the patient in the case under discussion.

On October 31, a boy, aged twelve years, living in the next street and attending the same school, was admitted to hospital with poliomyelitis; he died soon afterwards from respiratory involvement.

Discussion.

The clinical and other evidence in this case is consistent with the diagnosis of acute anterior poliomyelitis involving the lumbar enlargement of the spinal cord and a portion of the bulb, and it is not unreasonable to suggest that symptoms and signs were precipitated by a dental procedure. The interval between the trauma of injection and extractions and the development of symptoms and signs is difficult to fix. The mother seems quite certain that fever and marked drowsiness commenced on the day following dental treatment, but the precise day of onset of muscle weakness is not clear. It was probably not less than ten days after this treatment.

The local anæsthetic was deposited in tissue innervated by the trigeminal and facial nerves, while both the injection and the extractions must have been accompanied by some trauma of tissue supplied by these nerves.

McCloskey (1950), Geffen (1950) and Hill and Knowelden (1950) have established the role of injections in predisposing the subject to the onset of poliomyelitic paralysis, while Horstmann (1950), Harrington (1951) and Simpson (1951) have indicated an association between trauma and poliomyelitic paralysis.

There is no record of trigeminal involvement in the clinical notes appertaining to this patient, but trismus in poliomyelitis is usually attributed to irritation of the fifth nerve nucleus.

Bell's palsy is said to be a rare complication of local anæsthesia for a dental procedure and is usually of brief duration. It has been attributed by some to the effect of the anæsthetic upon the facial nerve itself. However, Stoy (1951) describes a case in which the paralysis lasted six weeks, and refers to an alternative mechanism indicated by the work of Kettel (1947), which suggests that the paralysis may be due to a reflex ischæmia in the vicinity of the stylomastoid foramen which results in œdema of the nerve within the bony canal and facial paralysis of relatively long duration.

The facial paralysis which may accompany up to 53% of cases of bulbar poliomyelitis (Baker, 1949), and which has been seen in bulbar cases in Perth, may be partial, patchy or complete. It may involve the upper part of the face, the lower part, or both; it does not necessarily spare the *orbicularis oculi*. It may be indistinguishable, on inspection, from classical Bell's palsy. On the whole it is believed to have a more favourable prognosis than the last-named disease, but in any event it may take many months to clear up, and even then may not do so completely. In this case, despite the lapse of three months, hemifacial weakness is still obvious.

Acknowledgement.

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Reviews.

DISEASES OF THE EAR, NOSE AND THROAT.

IN "Diseases of the Ear, Nose and Throat: A Textbook of Clinical and Laboratory Procedures", Georges Portmann, Professor of Oto-rhino-laryngology at the Bordeaux University, aims at describing with precision all the procedures of clinical examination of the ear, nose and throat patient.[1] He furthermore urges the surgeon to develop his power of observation, his diagnostic sense, and his psychological approach in taking the patient's history.

For each organ in the specialty, the anatomy, histology and physiology are dealt with and then the pathological symptoms and signs, together with the appropriate methods of history-taking and ferreting out of clinical signs in each case, are described. This is followed by mention of every conceivable clinical examination which might be of use, from the simplest to the most complicated. Radiological technique throughout is also given in a good deal of detail as well as radiographic and radioscopic interpretation. In fact, great detail and thoroughness are features throughout the whole big volume of more than seven hundred pages. The examination of static and dynamic equilibration alone occupies no less than 76 pages and 62 figures. It is in this section in particular that is keenly felt the total lack of references in the text to the appropriate figures, although in other parts of the book a very few of these references are made. Some of the tests described are most unusual and chiefly of academic interest, for example, anterior and posterior rhinomanometry. Professor Portmann's repeated

[1] "Diseases of the Ear, Nose and Throat: A Textbook of Clinical and Laboratory Procedures", by Georges Portmann, M.D., translated by Fernand Montreuil, M.D., and Jules G. Waltner, M.D.; 1951. Baltimore: The Williams and Wilkins Company. Sydney: Angus and Robertson, Limited. 10" x 7¼", pp. 736, with 11 plates in colour and 666 illustrations. Price: £10 15s.

statement that the larynx ends at the upper border of the first dorsal vertebra is not quite in accordance with English and American teaching.

The translation into English is bad, often clumsy, and at times is even carelessly done. For example, throughout the text the auditory apparatus is referred to as the auricular apparatus and the word "deformation" is consistently used when "deformity" is meant. "Female genital life" is another example of misappropriation of words, and on page 276 "myositis" should read "myosis". On page 477, it is correctly stated that the posterior crico-arytenoid muscle pulls the vocal process of the arytenoid laterally and on page 493 its pull on the process is medially. On page 486 the trachea bifurcates at the level of the fifth dorsal vertebra, but on page 491 it does so at the level of the third.

The impression is gained that the work of Professor Portmann, one of the world's most eminent oto-rhino-laryngologists, has been well and faithfully carried out in profound detail, but its value has been marred by somewhat inefficient translators who soon wearied in their long task. Nevertheless, in spite of these criticisms, the volume is still a valuable book for the student studying for a higher degree or diploma and is worthy of a place as a reference book on the shelves of the oto-rhino-laryngologist's library.

ANKYLOSING SPONDYLITIS.

THE appearance of the monograph "Ankylosing Spondylitis", by J. Forestier and F. Jacqueline, of Aix-les-Bains, and J. Rotés-Querol, of Barcelona, has been long awaited by rheumatologists.[1] The wide clinical experience of the authors has been fully employed in the production of a masterly treatise. Forestier has been well known for twenty years as a prolific writer on ankylosing spondylitis, and it comes as a surprise to find that this is his first monograph of book length on the subject. It is based on the study of more than 400 patients, most of them followed for a number of years and 40 of them for more than twenty years.

The authors have considered in detail the course of ankylosing spondylitis in a large number of cases. They have followed the development of the disease in the spine and all other joints which may be involved. The work is set out in four parts, as follows: Part I: General remarks, including history. Part II: Clinical aspects, including analysis of symptoms, modes of onset, course of disease, clinical variations, diagnostic problems and ætiology. Part III: Morbid anatomy and pathogenesis. Part IV: Treatment, including posture and preventive orthopædics, radiotherapy, gold salts, cortisone, heat, corrective orthopædics, and surgical operations. The bibliography contains 221 references. Unfortunately an adequate index is not provided.

The story has been told in an interesting fashion, and the manner in which it is set out has contributed to this end. Relevant case histories are not separated from the main text, but the general descriptions are repeatedly interrupted in order to make way for case records and personal experiences in smaller type. This prevents the work from ever becoming prosy.

The radiology is of the highest quality, something like 200 illustrative X-ray pictures being interspersed through the text, with a brief case history attached to each. The wide experience of Dr. J. J. Herbert, of Aix-les-Bains, has been called on in the discussion of corrective surgical procedures. The authors find that the effect of cortisone on radicular pains is less spectacular than on involved peripheral joints. Generally they have had the same results with cortisone as have many other authors. They recommend gold salts chiefly for patients with involvement of peripheral joints, and they believe that radiotherapy applied to the spine is of value.

The cause of ankylosing spondylitis remains unknown; the authors mention a dozen precipitating causes, but point out that the majority of cases start without apparent cause. In the inevitable discussion on the relation of ankylosing spondylitis to rheumatoid arthritis, the authors conclude that they are different clinical entities, and they frown on the use of the term "rheumatoid spondylitis". While they base sharp divisions on differing clinical pictures, however, one feels that they overlook essential similarities in the pathological picture.

Original clinical observations on any of the many aspects of rheumatic disease are of value, and always will be until its pathology is better understood. Perhaps the pathology will not be properly comprehended until its natural history has been written fully; and unfortunately there still remain large gaps in the natural history of rheumatic disease. There will soon come a stage at which further advance will depend on the correlation of facts already known rather than on the acquiring of new facts; and it will be through such contributions as this that the gaps in our knowledge will be filled.

[1] *"La spondylarthrite ankylosante: clinique, radiologie, anatomie pathologique, traitement"*, by J. Forestier, F. Jacqueline and J. Rotés-Querol; 1951. Paris: Masson et Cie. 10" × 7", pp. 334, with 143 illustrations. Price: 2650 Fr.

Books Received.

[The mention of a book in this column does not imply that no review will appear in a subsequent issue.]

"Topics in Physical Chemistry: A Supplementary Text for Students of Medicine", by W. Mansfield Clark, Ph.D., Sc.D.; Second Edition; 1952. Baltimore: The Williams and Wilkins Company. Sydney: Angus and Robertson, Limited. 9" × 6½", pp. 794, with many text figures. Price: £5 7s. 6d.

For medical students.

"Surgical Treatment of the Motor-Skeletal System", supervising editor, Frederic W. Bancroft, A.B., M.D., F.A.C.S., associate editor, Henry C. Marble, A.B., M.D., F.A.C.S.; Second Edition; 1951. Philadelphia, London, Montreal: J. B. Lippincott Company. Sydney: Angus and Robertson, Limited. In two volumes. Part 1: Deformities, Paralytic Disorders, Muscles, Tendons, Bursæ, New Growths, Diseases of Bones and Joints, Amputations. Part 2: Fractures, Dislocations, Sprains, Muscle and Tendon Injuries, Birth Injuries. 10" × 7½", pp. 1300, with 1049 illustrations and three colour plates. Price: £12 18s.

The material is presented from the standpoint of "indicating what to do, when to do it, how to do it, and what not to do" for the various conditions affecting the motor-skeletal system.

"Monographs on Surgery, 1952", edited by B. Noland Carter, M.D., Ph.D. Advisory editors: Gynæcology, Joe V. Meigs, M.D.; Urology, Charles Huggins, M.D.; Orthopædic Surgery, Alfred R. Shands, M.D.; 1952. Baltimore: The Williams and Wilkins Company. Sydney: Angus and Robertson, Limited. 10" × 7½", pp. 442, with 150 illustrations. Price: £7 14s. 3d.

The third volume of this series, which has replaced "Nelson's Loose Leaf Surgery".

"The Child in Health and Disease: A Textbook for Students and Practitioners of Medicine", by Clifford G. Grulee, M.D., and R. Cannon Eley, M.D.; Second Edition; 1952. Baltimore: The Williams and Wilkins Company. Sydney: Angus and Robertson, Limited. 10" × 7½", pp. 1250, with many illustrations, a few in colour. Price: £8 1s. 3d.

A complete revision with the addition of some new subjects.

"A Handbook of Operative Surgery: Surgical Gynecology: Including Important Obstetric Operations", by J. P. Greenhill, M.D.; 1952. Chicago: The Year Book Publishers, Incorporated. 8½" × 6", pp. 350, with 101 illustrations. Price: $8.50.

For young gynæcologists, general surgeons and those physicians in general practice who also perform operations.

"Physiological Changes in Plasma Proteins Characteristic of Human Reproduction: Cross-Sectional and Longitudinal Electrophoretic Data for Women during and following Uncomplicated and Complicated Pregnancies", by Icie G. Macy, Ph.D., Sc.D., and Harold C. Mack, M.D. Detroit: Children's Fund of Michigan. 8½" × 6", pp. 178, with one plate and fourteen text figures. *Gratis.*

The results of electrophoretic studies of blood plasma proteins.

"Modern Trends in Gastro-Enterology", edited by F. Avery Jones, M.D., F.R.C.P.; 1952. London: Butterworth and Company (Publishers), Limited. 10" × 7", pp. 844, with 275 illustrations. Price: £7 7s. 6d.

Recent work in the gastro-enterological aspects of medicine and surgery, discussed by appropriate authorities.

The Medical Journal of Australia

SATURDAY, JULY 5, 1952.

All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given without abbreviation: surname of author, initials of author, year, full title of article, name of journal without abbreviation, volume, number of first page of the article. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors who are not accustomed to preparing drawings or photographic prints for reproduction are invited to seek the advice of the Editor.

POLIOMYELITIS.

If a medical officer of public health was asked to name the disease which held the interest and captured the imagination of medical practitioners and the non-medical members of the community more than any other, he would probably without hesitation name acute anterior poliomyelitis, or as many people call it, infantile paralysis. This is easy to understand from the point of view of the layman because of the suddenness with which apparently healthy people may be affected and the obvious and severe sequelæ with which they are left for all time. The medical observer cannot fail to be impressed by the characteristics of the disease, as the layman is, but he also has presented to him problems of first-rate importance and of peculiar intricacy. The disease is suited in many ways to experimental investigation and workers in many places have made the most of their opportunities; so much so that we have reason to expect before long the perfection by the laboratory worker of specific means of diagnosis and treatment. It is interesting to look back on the progress of knowledge over the years and to see how some concepts previously held have developed and others have had to be modified. In THE MEDICAL JOURNAL OF AUSTRALIA of September 1, 1928, will be found a symposium on poliomyelitis based on lectures and demonstrations organized by the Melbourne Permanent Committee for Post-Graduate Work. The introductory paper on epidemiology was presented by John Dale and covered the subject in a comprehensive fashion. Reginald Webster gave a demonstration of the pathological aspects of the disease. F. G. Morgan discussed the preparation of serum from human donors recovered from poliomyelitis. Jean Macnamara discussed the serum therapy of the disease and its treatment during the acute and convalescent stages. H. C. Trumble dealt with the construction of plaster moulds, F. Kingsley Norris with splinting in acute stages and W. Kent Hughes with treatment in the acute stages. Reginald Webster in the course of his demonstration discussed the causative virus and pointed out that the clinical and anatomical changes in the experimental disease very closely paralleled those in the human subject. He stated that in the spontaneous disease in human subjects the virus, having reached the upper respiratory mucous membrane, multiplied locally until a point was reached at which it invaded the bloodstream. At the same time mention was made by another speaker of the fact that milk could carry the infection. The respiratory tract was accepted for many years as the portal of entry of the virus. In Topley and Wilson's "Principles of Bacteriology and Immunity", second edition (1936), it is stated that infection most probably occurs by the respiratory tract. At the same time it is pointed out that infection by the gastro-intestinal tract has been suggested since the occurrence of occasional outbreaks caused by milk. Experimental evidence by Flexner is stated not to support this view, and infection by this means is thought to be uncommon. We now know that infection by way of the intestine is generally accepted by present-day investigators.

In this issue we publish some of the papers read at a symposium arranged by the Melbourne Permanent Post-Graduate Committee and the Consultative Council for Poliomyelitis of Victoria together with some papers received from other sources. The symposium papers, which we publish, by Jean Macnamara, H. McLorinan, Stanley Williams and Bertram P. McClosky, deal with the clinical and public health aspects of the subject. Unfortunately the papers which were presented on what we may call the laboratory side were not made available for publication; it is hoped that they may be published in another form at a later date. The paper by Ethel Byrne and Alan T. Roberts is unusual and will no doubt give rise to some comment. Attention may be drawn to several points in it. In the first place their non-use of restraining splints does not agree with the statements by McLorinan and by Macnamara. The former points out that, though immediate splinting is rarely required, the use of sandbags, pillows, slings and plaster casts is necessary to avoid malformation and subsequent development of deformities; and that, usually within a fortnight of the onset, some form of rigid splinting should be applied. The latter declares unequivocally that accurate, evaluated splinting is our most useful tool in the prevention and reduction of crippling following poliomyelitis. Byrne and Roberts's statements on respirators will arouse comment and most orthopædic surgeons will criticize their failure to use muscle charting; at the same time their paper presents a point of view which should not be overlooked.

It is on what we have called the laboratory side that most progress has been and is being made. A useful outline of recent work will be found in THE MEDICAL JOURNAL OF AUSTRALIA of December 8, 1951, in a report of the second International Poliomyelitis Conference held at Copenhagen last September. F. M. Burnet also made a clear and concise statement on the problems of poliomyelitis in the course of a paper on poliomyelitis and Murray Valley encephalitis read to the New South Wales Branch of the British Medical Association and published in this journal on February 9, 1952. Much of the progress has been due to the tissue culture of poliomyelitis virus described by J. F. Enders, of Harvard. Enders used what is known as the roller tube technique based on a method

used by Zinsser for the cultivation of Rickettsia. It was by this means that the three strains—Lansing, Brunhilde and Leon—were isolated. Addition of serum containing the appropriate antibodies will prevent growth of a virus in the roller tube; thus cultures of known strains of virus can be used to test for the presence of antibody in a particular living patient. These observations have been used in the study of epidemics. But to return to our general story as told by Burnet at the Sydney meeting—the intestine, he remarked, was known to be the site of election for three poliomyelitis viruses, at least ten Coxsackie type viruses and almost certainly the virus of infectious hepatitis. He pointed out that enormous amounts of poliomyelitis virus were excreted in the fæces of any community in which poliomyelitis was active, and that there was a growing belief that poliomyelitis was spread predominantly by transfer of infected fæces in one fashion or another. At the same time he admitted that droplet infection could not be excluded and was favoured by some authorities. In regard to the regularities between the presence of poliomyelitis virus in the intestinal tract and the occurrence of paralytic disease, Burnet said that there were differences between strains in their capacity to infect the central nervous system, and these were called their invasiveness. Differences in the invasiveness of different strains were almost certainly the main reason for the difference in intensity of poliomyelitis epidemics in any community which had maintained a reasonably constant social structure. Burnet thinks that nearly all the difficulty of understanding the epidemiology of poliomyelitis disappears if one accepts the view that the proportion of individuals infected for the first time who suffer from paralytic infection varies characteristically with age, being lowest in infancy and reaching a maximum in adolescence and young adult life. Finally he knows of no facts to contradict the dual deductions (*a*) that the prevalence of poliomyelitis in advanced rather than in backward countries is due primarily to the relative absence of subclinical immunization in the earliest years of life, and (*b*) that the only logical approach to the prevention of poliomyelitis is the development of some means of artificial immunization which will effectively replace the natural process. It is immunization for which the whole scientific world is waiting and which, we are given to understand, is not so far off. Burnet thinks that the only visible approach is the use of a living non-virulent virus administered by the natural alimentary route of infection. If a strain completely innocuous to infants and with immunizing powers could be grown in large scale in tissue culture by the Enders method, it would, in Burnet's opinion, rapidly become a widely or universally used prophylactic. Speaking of large-scale immunization against poliomyelitis, Burnet used the following words: "When we realize that to be effective it must be applied in the first year or two of life and that there is no certain means of ensuring the harmlessness of a living poliomyelitis virus except actual test in infants, we can appreciate the courage (or foolhardiness) that will be needed."

One group of workers has been sufficiently courageous (or foolhardy) to do this thing. In the *American Journal of Hygiene* for January, 1952, they write on "Immune Responses in Human Volunteers upon Oral Administration of a Rodent-Adapted Strain of Poliomyelitis Virus". In an early paragraph of their paper, either by way of introduction or as justification of their efforts, they quote words of Burnet published in *The Bulletin of the Johns Hopkins Hospital* to the effect that sooner or later it will become necessary to use living virus vaccines given by mouth in infancy, perhaps under cover of γ globulin passive protection. The authors are Hilary Koprowski, George A. Jervis and Thomas W. Norton. They had twenty volunteers, but give no information about them "for obvious reasons"; the age of the volunteers at least would be important in this type of investigation. However, the volunteers were given by mouth a strain of rodent-adapted Lansing-like poliomyelitis virus. Of the 20 volunteers there were 15 whose serum was devoid of neutralizing power against the strain of virus used, and 12 of the 15 became carriers and their serum manifested a high concentration of antibodies. The three subjects who did not yield virus in their fæces were also found to have neutralizing antibodies. No signs of illness of any kind were observed among the subjects. The antibodies which appeared were type specific—in no instance were Brunhilde antibodies found. It is tempting to draw conclusions from this work, but Koprowski and his co-workers resist the temptation. They state that they must await proof that the procedure is really safe. Clearly further studies along these lines will be made, studies of the kind envisaged by Burnet in his Sydney paper. Koprowski and his collaborators regard their findings as "a first step in an attempt to transfer results obtained in the laboratory to actual field investigations". And that is what they are.

Current Comment.

SURGERY AND THE AGED PATIENT.

A LAYMAN not so long ago described the practice of surgery as a "ticklish business". At first one might ask who was tickled—the patient or the surgeon—and the idea might lead to peculiar flights of fancy. Recourse to the "Concise Oxford Dictionary", however, shows that "ticklish" has several meanings. It means not only "easily tickled", "sensitive to tickling" but also "difficult", "critical", "delicate", "requiring careful handling". When we see that "to tickle" means "to apply light touches" to a person to produce a certain result, we may accept the word ticklish in relation to surgery. The successful surgeon is one who treats the tissues of the body with respect, who applies light touches to them when he works with his scalpel or other implement. Of all the branches of general surgery operation on elderly persons may be regarded as the most ticklish. The tissues of the aged need more careful handling than those of any other group of persons, and we may say that a surgeon who is successful with his operations on old people has mastered one of the most important aspects of his craft. For this reason the surgery of old age is a subject to which repeated reference is justified.

In an article entitled "The Elderly Surgical Patient", D. T. Bosch, A. Islami, C. T. C. Tan and C. A. Beling[1] have made an analysis of 500 consecutive patients sixty years of age and over, who were subjected to major surgical operation between May 1, 1947, and December 11, 1949, at one hospital. No case was included twice in the list. When the same patient had undergone more than one major operation during the period of study, the procedure of the greatest magnitude was selected for inclusion. Of the 500 patients 44% had previously undergone one or

[1] *A.M.A. Archives of Surgery*, March, 1952.

more surgical operations. From this the authors draw an unjustified conclusion: "This would roughly indicate that in American metropolitan areas nearly half of the population will have been subjected to some form of major surgery by the time 60 years of age is reached." It is clearly not right to regard the 500 patients sixty years of age and over who came to hospital for major surgical operation as a typical cross-section of the same age period in the community at large. However, this conclusion has nothing to do with the general purpose of the paper. The hospital mortality rate for all major surgical procedures during the period covered was 3·01%, a surgical death being defined as "one which followed surgery regardless of cause or time between operation and death provided the patient was still in hospital". If palliative and purely exploratory operations were excluded, the mortality rate was 2·3%. For the series of 500 patients over fifty-nine years of age the mortality rate was 9·6%, or about three times the rate for all major surgical conditions. Patients seventy years and over numbered 159, and the death rate among these patients was 16·1%. The types of operation were what might be expected; totals over 20 were as follows: suprapubic prostatectomy, 46; cholecystectomy, 45; inguinal herniorrhaphy, 40; vaginal hysterectomy, 28; radical mastectomy, 23; exploratory laparotomy without further operation, 23; partial resection of the colon, 22; colostomy, 22; hip pinning, 22. The type of anæsthesia used had no effect on the mortality rate; most patients received cyclopropane (160), 124 were given nitrous oxide, oxygen and ether, 122 were given thiopental sodium, and only 19 were operated on under local anæsthesia. Bosch and his colleagues conclude that age alone should not contraindicate surgical operation. They suggest the following "improvements" in the management of elderly surgical patients. (i) An optimistic approach should be adopted. (ii) Since the presence of concurrent disease, such as a cardiac disorder, does not create an insurmountable risk, more cooperation between internist and surgeon should result in better management. (iii) Infection remains a problem regardless of antibiotic therapy, for elderly persons tolerate infection poorly. Therefore all precautions to avoid infection should be taken and specific antibiotic therapy instituted whenever possible. (iv) Quantitative repletion based on pre-operative blood-volume studies should be performed routinely.

This study calls to mind one made by the late Alan Newton and published in this journal on February 8, 1936. Incidentally we may remark that the American authors and Newton both quoted the verse from the ninetieth Psalm of David that: "The days of our years are three score years and ten; and if by reason of strength they be fourscore years, yet is their strength labour and sorrow; for it is soon cut off, and we fly away." Newton reported 100 operations performed on 98 patients over seventy years of age. His series included all patients in the eighth and ninth decades of life who had been referred to him during a stated period for operative treatment of a major type. This was thus an older group than that described by Bosch and his co-workers. The series included consecutive cases and in no instance was operation refused because of the severity either of the disease or of the operation required. The types of operation were much the same as those mentioned by the American authors. Eight only of the patients died—a remarkable result when we remember that Newton was writing in the pre-antibiotic era. He concluded, in much the same fashion as the American group, that "the age factor should not be given undue consideration when operative measures are necessary". We would suggest that present-day surgeons would profit from a careful study of Newton's article.

Attention may also be drawn to a contribution to the subject by J. W. S. Laidley in a paper read before the New South Wales Branch of the British Medical Association and published in this journal on January 31, 1948. Laidley drew attention to the post-operative complications and declared that every advance in anæsthesia made surgery in the aged safer. He also remarked that the surgeon had to employ all his wits before he made up his mind that surgery would really benefit his patient; once that decision was made he had to plan to inflict as little surgical trauma as possible. The general conclusion to be drawn from all these papers is that the surgeon must not shun the "ticklish" business of operating on old people, but rather look on the results obtained by him as an indication of his own efficiency.

FELLOWSHIP OF THE ROYAL SOCIETY.

AMONG the new Fellows of the Royal Society elected on March 20 of this year is Dr. M. R. Lemberg, director of the biochemical laboratories at the Institute of Medical Research, Royal North Shore Hospital of Sydney. Dr. Lemberg has published many papers on the subjects of blood and bile pigments and hæmoglobin metabolism. Graduating as Ph.D. at the University of Breslau, he became a lecturer at the University of Heidelberg. He went to Cambridge as a Rockefeller Foundation Fellow, and there worked under the late Sir Frederick Gowland Hopkins and also with Barcroft and Keilin. While at Cambridge in 1935, on the recommendation of Hopkins, he was invited to take up his present position at the Royal North Shore Hospital of Sydney. In 1949, at the invitation of the publishers, and in collaboration with J. W. Legge, he published a book in the Interscience Series entitled "Hæmatin Compounds and Bile Pigments, Their Constitution, Metabolism and Function". Dr. Lemberg is a member of the Advisory Research Committee of the National Health and Medical Research Council.

Another of the new Fellows who will be known to many in Australia is Professor Bernard Katz, who is professor of biophysics at University College, London. Professor Katz was for a time a member of the staff of the Kanematsu Memorial Institute of Pathology, Sydney Hospital, and an honorary lecturer in physiology at the University of Sydney. His most notable work has been in the field of neurophysiology.

On behalf of the medical profession of Australia we offer Dr. Lemberg and Professor Katz congratulations on their well-deserved honours.

A MEMORIAL TO THE LATE FRANK SANDLAND HONE.

THE Council of the South Australian Branch of the British Medical Association decided a month or two ago to establish a fund to commemorate the work of the late Dr. Frank Sandland Hone. Already the response in South Australia has been good, many contributions having come from old patients and non-medical friends. Hone's work, however, was not confined to South Australia. He gave of his profound knowledge, of his time and his energy to activities of the British Medical Association in the Federal sphere, to the University of Adelaide, to the School of Public Health and Tropical Medicine established at the University of Sydney by the Commonwealth Government, to THE MEDICAL JOURNAL OF AUSTRALIA and the Australasian Medical Publishing Company, Limited, to the Royal Commission on Health in the 'twenties, and to other undertakings calculated to advance the science and art of medicine in this country. Many members of the medical profession will wish to share in the creation of a memorial worthy of the man and his work. Dr. R. L. Thorold Grant, the President of the South Australian Branch, states that the fund is open for subscription by all members of the profession. The object of the appeal is twofold: (*a*) to endow a Frank S. Hone Memorial Prize in Medicine at the University of Adelaide in connexion with the medical group of subjects in the final examination; (*b*) to institute in perpetuity a Frank S. Hone Memorial Oration dealing with the relation between medicine and the community. Subscriptions may be sent to the Secretary, South Australian Branch, 178 North Terrace, Adelaide.

Abstracts from Medical Literature.

RADIOLOGY.

Intervertebral Joint and Foramen Abnormalities.

LEE A. HADLEY (*Radiology*, March, 1951) states that the anatomy of the intervertebral foramen allows a generous reserve safety cushion space about the nerve root. Within this space occur the compressible lymphatics, blood vessels, and areolar and fatty tissue. The normal movements of the spine may produce a physiological constriction of the foramen amounting to as much as a third of its area. This explains the positions assumed by patients suffering from nerve root pressure and the reason for aggravation of their pain upon certain movements. Permanent constriction of the foramen from any cause reduces the reserve cushion space and predisposes to root pressure upon occurrence of œdema, hæmorrhage, perineural inflammation or additional foramen encroachment. Foramen constriction may result from various causes, but the most common is disk degeneration and its secondary changes; these include herniation of disk substance into the foramen, thinning of the disk with approximation of the pedicles, subluxation of the upper vertebræ forward or backward upon the one beneath, hyperplasia of the *ligamentum flavum*, bony spur formation projecting backward from the disk or forward from the posterior joint, and, lastly, subluxation of the articular process from below upward and forward into the foramen. Bony impingement of the tip of the articular process may occur with development of erosion and sclerosis at the point of contact. Nerve roots removed from constricted foramina have shown definite degenerative changes in their morphology.

X-Ray Findings Accompanying Carcinoma of the Pancreas.

J. W. BEELER AND B. R. KIRKLIN (*American Journal of Roentgenology*, April, 1952) state that with the recent encouraging note in the results of radical pancreato-duodenectomy for carcinoma of the pancreas, the focus of attention is on the earliest possible pre-operative diagnosis. It is well known that the mass of pancreatic carcinoma is responsible for certain X-ray findings, but it is less widely known that actual invasion of the stomach or duodenum plays as important a part in production of positive X-ray findings. It is important to note that in many of the cases in which the X-ray findings were positive, the tumours were very small, 57% being less than six centimetres in diameter. Frequently, therefore, it is a combination of the two factors, mass and invasion, which produces the alteration. The first portion of the duodenum was the most frequent location to show these changes, the first and second portions together were the next most common site, and the second portion alone was the third most common site. It is the duodenum, therefore, that particularly should be watched in any suspected case of pancreatic malignant disease. The carcinoma may be of sufficient size to be palpated in the course of fluoroscopic examination, and because of its mass it may distort, obstruct, narrow or produce a filling defect in any portion of the stomach or duodenum, depending on the site of origin of the carcinoma in the pancreas. In only 16% of cases was the "typical" widening of the duodenal loop seen, indicating that it is not a frequent finding in the condition. When the head of the pancreas is the site of the tumour, the close proximity of the duodenum leads to invasion or pressure deformities much more readily than when the lesion is in the body or tail. Usually a tumour in either of these latter sites must be large if it is to make its presence evident in examination of the stomach.

Fracture of the Spinous Processes.

PETER ZANCA AND ELMER A. LODMELL (*Radiology*, March, 1951) state that patients who have received a severe head or neck injury usually report to the hospital with the head held either in hyperflexion or in hyperextension. On the routine lateral view of the neck it is difficult to see the spinous processes of the seventh cervical and first thoracic vertebræ because of the superimposition of the shadows produced by the bone and soft tissues of the shoulder girdle. Under such circumstances, the antero-posterior view of the spine should be sufficient to make a diagnosis of fracture. The radiographic diagnosis of fractures of the spinous processes of the seventh cervical and first thoracic vertebræ can be made on the preliminary antero-posterior view of the cervical and upper thoracic parts of the spine. The fracture can be identified by the malalignment and downward displacement of the outer end of the spinous process. On the X-ray film, a double shadow is seen. A smooth, crescentic area of decreased density represents the fractured base of the spinous process, and a denser shadow lying in the soft tissues, slightly caudad to the base of the spinous process, represents the displaced tip.

Air-Contrast Study of the Duodenal Bulb.

RALPH R. MEYER (*Radiology*, March, 1952) describes the technique for air-contrast study of the duodenal bulb and emphasizes its value in demonstrating an ulcer crater, particularly when compression of the bulb is to be avoided because of recent gross gastro-intestinal hæmorrhage, or when the bulb is inaccessible to compression. Air-contrast study of the duodenal bulb is regularly performed after the routine examination of the patient in the upright position. After the bulb has been filled with barium and compression attempted, the patient is placed supine. In this position the air normally present in the gastric fundus will gravitate to the antral end of the stomach. The bulb fills with air when it is elevated above the level of the stomach by raising of the patient's right side off the table. The degree of obliquity depends on the position of the bulb. A high oblique or lateral position is necessary to outline a bulb directed posteriorly; a slight oblique position will suffice when the bulb lies in the transverse plane. Air will usually fill the bulb in a few seconds, a thin coating of barium being left on the mucosa. When filling is not prompt, compression of the antrum with the gloved hand or having the patient lie on the left side for a minute may be helpful. Spot films are routinely made because fluoroscopic visualization of the air-filled bulb is poor.

Osseous Lesions of Tuberous Sclerosis.

JOHN F. HOLT AND WILLARD W. DICKERSON (*Radiology*, January, 1952) state that from a purely objective radiological point of view the cyst-like lesions of the phalanges in tuberous sclerosis closely resemble the osseous lesions of sarcoidosis, but certain differential features are evident. The fine reticular pattern of bone destruction so characteristic of sarcoid is not found in tuberous sclerosis; there is less distortion of the general trabecular pattern in localized sarcoid lesions, and, above all, sarcoidosis almost never produces associated periosteal thickening. Entire phalanges may be destroyed without the slightest evidence of subperiosteal new-bone formation. Furthermore, when phalangeal lesions are found in tuberous sclerosis, cranial plaques or intracranial calcification, or both, will almost invariably be present. Such cranial abnormalities do not ordinarily occur in sarcoidosis. The degenerative cyst-like lesions commonly associated with the chronic arthritides might conceivably be confused with the phalangeal lesions of tuberous sclerosis, but associated joint changes in the former should serve to differentiate them. Diffuse, uniform, reactive periosteal new-bone formation of the metatarsals occurs frequently in patients with localized disorders of the feet due to arch defects, faulty weight-bearing *et cetera*. It should be emphasized, however, that the periosteal thickening in tuberous sclerosis is more often of a localized nature or, if extensive, of an irregular wavy variety. Low-grade inflammatory lesions of the metatarsals may simulate the periosteal thickening of tuberous sclerosis, but other radiological as well as clinical signs of an infectious process will serve to make the differentiation.

PHYSICAL THERAPY.

Soft-Tissue Technique in the Diagnosis and Treatment of Head and Neck Tumours.

G. H. FLETCHER AND K. E. MATZINGER (*Radiology*, September, 1951) state that irradiation of malignant neoplasms of the head and neck requires an accurate knowledge of the point of origin and extent of the tumour. The usual clinical methods of investigation often fail to reveal the true extent of the lesion, and in some instances, for example, in the naso-pharynx, the most careful examination often fails to reveal the primary lesion. Soft tissue films may give information not obtainable by other means, for example, diagnosis of a tumour which escapes clinical observation, determination of the extent and clinical variety, aid in planning X-ray treatment, and evaluation of results of treatment by changes in the films. Lateral and antero-posterior soft tissue films are essential, as well as special views for special sites. For the naso-pharynx, after instillation of lipiodol in both nares, a film is taken with the patient lying on his back in

hyperextension, the head resting against the cassette holder. For the laryngopharynx, after the ingestion of barium, tomographs may also be very helpful. The depth of the layer is usually selected from the lateral soft tissue film, the Adam's apple being used as a point of reference. The authors describe the techniques thought desirable for tumours in naso-pharynx, oropharynx, laryngohypopharynx, cervical part of the œsophagus and thyroid.

Treatment of Ovarian Carcinoma.

H. E. SCHMITZ AND J. T. MAJEWSKI (*Radiology*, December, 1951) analyse a group of 143 cases of ovarian carcinoma in which observation has been carried on for at least five years after irradiation. They state that the onset is frequently insidious in this disease, and the early stages are unrecognized by both the patient and the physician. In this group the peak of the age incidence was in the sixth decade, though there were two patients under twenty years of age. Of the 143 patients, 105 (73·4%) received beneficial extirpating surgery. In 16 cases (approximately 11%) an exploratory procedure was performed; in 13·2% no surgical intervention was made, because the patient could not stand the procedure or was beyond surgical aid. The authors hold that when ovarian malignant disease is suspected at operation, radical and extensive surgical procedures should be carried out. They advocate total hysterectomy with bilateral oophorectomy. The policy followed in this series was to extirpate as much diseased tissue as possible and to follow this with deep X-ray therapy. X rays generated at 800 kilovolts with a half-value layer of three millimetres of copper were used, and a total tumour dose of from 4500*r* to 5000*r* was given. The five-year survival rate in this group of patients was 20·28%. When all cases are eliminated which had progressed beyond the stage at which complete surgical removal of the grossly diseased tissue was possible, the survival rate is approximately 50%. The results compare favourably with those of others reported in the literature and emphasize the value of post-operative irradiation.

Radiation Hazards to the Embryo and Fœtus.

L. B. RUSSELL AND W. L. RUSSELL (*Radiology*, March, 1952) have conducted an extended series of experiments on the radiosensitivity of the embryo and fœtus and give some practical recommendations which, in their opinion, may protect the well-being of many unborn infants in this age of the increasing use of radiation. They have reached the following conclusions. The developing embryo of a great variety of animals studied is highly susceptible to the induction of malformations by irradiation; there is no reason to doubt that this also applies to human embryos. There are well-defined critical periods in the development of most characters, and in mice the periods correspond to the period in man from the second to the sixth week of gestation. Irradiation at more advanced stages produces less obvious and possibly more delayed effects. Doses high enough to produce developmental abnormalities do not necessarily cause abortion or pre-natal death. The major malformations are probably due to direct radiation effect on the embryo rather than to indirect action via maternal injury. Doses as low as 25*r* have been shown to be effective in producing particular changes if applied at the critical time, and it is quite possible that lower doses well within the range of diagnostic fluoroscopy may cause developmental alterations which may be important in the human. The authors recommend that, whenever possible, irradiation of the uterus in women of the child-bearing age should be restricted to the two weeks following the last menstrual period to preclude the possibility that fertilization may have taken place; this applies to diagnostic irradiation even if the dose involved is less than 25*r*. The practice of avoiding irradiation during a known pregnancy should be continued.

Thymic Tumour, Myasthenia Gravis and X-Ray Therapy.

I. G. WILLIAMS (*Journal of the Faculty of Radiologists*, January, 1952) states that *myasthenia gravis* is associated, in some way which at present is not understood, with the thymus gland. The work of Keynes, in England, and Blalock and Veits, in America, has proved beyond doubt that removal of the thymus results in relief of the myasthenia in many cases. The treatment of *myasthenia gravis* falls into two large groups: (i) Attempted restoration of the balance between acetylcholine and cholinesterase by the administration of drugs which act on the myoneural junctions; in this regard, ephedrine and neostigmine are the only substances which act over a long period. (ii) Abolition of thymic activity related in some unknown fashion to these myoneural reactions; ablation of thymic activity may be achieved by surgical removal of the gland, or by the destruction of essential cells by means of irradiation. Keynes had up to September, 1949, operated on 155 patients including 11% with tumours. Of 120 patients without tumours, 79 (62%) had a complete remission of symptoms. It is said that in the presence of tumours the prognosis is very much worse. Although immediate response may be good, the tumour may recur locally or within the thoracic cage. For these reasons, patients are referred for radiotherapy. The author records his experiences with 13 cases of thymic tumour associated with *myasthenia gravis*. He states that radiotherapy may be given in three ways: (*a*) As a pre-operative measure. The patient may be made safer for surgery by lessening the severity of the myasthenia and the operation made easier technically because the tumour is smaller. (*b*) As a post-operative measure. This is regarded as irrational, and the advantages are said to lie with pre-operative radiotherapy. (*c*) As a possible method of treatment by itself. This is the subject of an investigation which is still proceeding; the rationale of radiotherapy is based on the histology of the thymus and of the tumour, both of which are inherently radiosensitive. The author states that X-ray therapy has been used in all cases. At first the conventional 250 kilovolt deep X-ray therapy was used, but now the treatment is at 1,000,000 volts in all cases. The initial doses are kept small, 50*r* or 100*r* being given to the tumour daily. Too large a dose at first may increase the severity of the myasthenia. The limit has been set at a tumour dose of 4000*r* in four weeks. Of the nine patients treated in association with surgery, eight are alive, one after three years, one after two and a half years, and one after two years. Four patients had irradiation alone. One with severe disease and generalized myasthenia dispensed with neostigmine in six months and has led a normal life for two and a half years.

Radiation Histology.

G. GRICOUROFF (*The British Journal of Radiology*, January, 1952) states that it would be particularly important, in the radiotherapy of cancer, to know how and in what way a histological study would enable a prognosis to be given in relation not only to local sterilization of the cancer, but also to the ultimate recovery of the patient. He asks the question whether viable cancer cells can escape destruction after exposure to a homogeneous dose sufficient to cause clinical disappearance of the tumour, and concludes that failure to sterilize a cancer may be due to the persistence of neoplastic cells which are not specifically different from others but are momentarily in some particular phase of activity. A very few uninjured cells (theoretically only one cell) will be sufficient to start the cancer growing again.

A. GLÜCKSMANN (*ibidem*) states that penetrating radiations can, under certain circumstances and in certain normal and malignant human tissues, change the type of differentiation or affect its extent and degree. By no means all tumours respond to irradiation with increased differentiation. Many are locally incurable by irradiation, with doses within the present therapeutic range, and show only transitory decreases in mitotic activity and increases in cell deaths as the result of both direct and indirect irradiation effects, but no obvious changes in extent and degree of differentiation. The histological changes in radio-curable epitheliomata on the other hand are very rapid and pronounced and very frequently characterized by striking increases in the degree and extent of differentiation in addition to the other effects of radiation. Observations on serial biopsies from epitheliomata taken at various stages during treatment have revealed that most of the resting cells are not killed outright by radiation, but are able to undergo abnormal mitosis, to enlarge, to form prickles and keratin, and thus to sterilize themselves by differentiation. Cure by irradiation of rodent ulcer, of uterine adenocarcinoma and of a variety of breast cancers cannot be attributable to differentiation, since these tissues are not capable of post-mitotic differentiation. The author records the correlation of local histological prognosis with the local clinical result of radiotherapy five to twelve years after treatment in 325 cases, and shows that the prognosis given was correct in 95% of cases (either favourable or unfavourable). He states that the mechanism by which irradiation increases the relative extent of differentiation may be related to the injury of chromosomes, which results in pronounced nuclear abnormalities and, while allowing the basal cells to differentiate, prevents them from undergoing division.

Special Articles for the Clinician.

(CONTRIBUTED BY REQUEST.)

XXVII.

THE ÆTIOLOGY OF ANÆMIA.

OUR teeming billions of red blood cells, constantly circulating, form a device, ingenious beyond compare, with which Nature and evolution have endowed the higher vertebrates for the supply of oxygen and removal of carbon dioxide so essential for the physiological performance of all organs and tissues, including those responsible for blood formation. To be fully effective in their "community service", the red cells must be normal in numbers, hæmoglobin content and morphology.

In health, this normality of red cells is maintained by a sequence of: (*a*) dietary sufficiency of substances necessary for erythropoiesis; (*b*) conversion, by gastro-intestinal activity, of the ingested "building stones" into chemical forms suitable for absorption, storage and utilization; (*c*) a balance between the rate at which effete red cells break down and are removed from the circulation and that at which new cells are concurrently formed; and (*d*) equilibrium between the demands made by special or periodic physiological stresses and compensatory erythropoietic activity.

Defects at any point or points in this sequence may lead to anæmia, which is commonly classified correspondingly into four main types—nutritional, dyspoietic, hæmolytic and hæmorrhagic. Although the processes in the sequence are largely interdependent, characteristic numerical and morphological abnormalities arise in the red cells, which are determined, not only by the nature of the defect, but also by the point in the sequence predominantly at fault.

The present-day concept is therefore that, even in well-defined syndromes of which anæmia is a prominent feature, its existence is, with rare exceptions, due to identifiable underlying ætiological factors, of which it must be regarded as symptomatic evidence, rather than as a disease entity in itself.

Irrespective of signs or symptoms directly referable to the causal defect, anæmia *per se* does, however, bring further well-known symptoms in its train, the severity of which increases *pari passu* with that of the anæmia; for this reason, the earlier concept of anæmia as a primary disease might seem logical. We would not, however, be satisfied with a diagnosis of "pressure" when this condition is present as a result of a tumour mass.

Moreover, the symptoms associated with the anæmia rather than those of its cause are sometimes the first to direct a patient's attention to his lack of well-being. They may thus initially dominate the clinical picture when he seeks medical advice. It is all the more important therefore to appreciate the symptomatic significance of anæmia, and, after establishing its type by blood examination, to identify, and whenever possible to remove or cure the causal abnormality. This course of action should not exclude simultaneous attempts to relieve an anæmic patient's symptomatic distress by correct hæmatinic treatment, although such treatment may be less than maximally effective when underlying disease is present.

It is therefore usually bad medical practice to give hæmatinic treatment without attempted control of the underlying defect. Such practice may lead to long or unsuccessful courses of treatment which are fundamentally uneconomic, even if "free" to the individual patient. More harmful is the risk of deferred discovery of neoplastic or other disease, for which early treatment is important, or of a seriously wrong prognosis. Occasional patients can be recalled who, after unsuccessful empirical treatment for varying periods for anæmia alone, were found when subsequently referred for hæmatological investigation to be suffering from such conditions as carcinoma, peptic ulcer, intestinal parasitic infestation, hæmorrhoids, subacute bacterial endocarditis, nephritis, leuchæmia or uterine fibroid tumours.

It may be difficult to decide, on clinical evidence, whether or not anæmia is present (sun tan and cosmetics alone are great deceivers), but after its existence has been proven, unless ætiological factors are obvious, consideration of all possible causes should be systematically planned. The classification of anæmia into the four main types mentioned above, though hæmatologically sound, may not provide the most practical approach for the clinician faced with the problem of identifying the cause of the anæmia in an individual patient. After noting the patient's age, sex, occupation, medical history, and the results of initial clinical and laboratory investigations (which may suggest the probable ætiology and further tests likely to be of value), he may prefer to consider possible causes of the anæmia under the following headings:

I. Dietary deficiencies.

II. Causes arising within the body:
- (*a*) Special stresses of physiological origin.
- (*b*) Disease or dysfunction.
- (*c*) Constitutional or hereditary defects.
- (*d*) Intake of harmful chemical or physical agents.

III. Causes which cannot be identified.

DIETARY DEFICIENCIES.

Iron.

The importance of iron as one of the chief components of hæmoglobin needs no stressing. Food iron can be absorbed, and hence become available for hæmoglobin synthesis, only if it is convertible by digestion into the ionic (preferably ferrous) state. The convertibility of iron varies in different foods, and in this respect red meat, though of great value as a source of protein and some vitamins, falls short of some other foods in respect of the availability of its abundant iron. Foods with useful amounts of iron include oatmeal, wholemeal bread, liver, eggs, green and other coloured vegetables, many dried fruits, cocoa and chocolate. Iron once absorbed, or when liberated from effete red cells, is, however, almost entirely conserved within the body, and can be used for hæmoglobin synthesis. It has been estimated that reasonable amounts of the foods listed above will be ample to meet the requirements of men, but provide only a very small safety margin for women and growing children, since pregnancy and lactation, menorrhagia, or rapid growth may deplete the iron reserves. Iron deficiency anæmia is characterized by hypochromia, with or without other changes in the red cells.

Other metallic elements, including copper and cobalt, are adjuvants to iron, but, in man, are needed in such minute traces that dietary deficiencies are unlikely.

Phosphorus in excess may combine with ingested iron to form insoluble, and hence unabsorbable, iron phosphate, an effect which is counteracted by moderate amounts of calcium, but excess of the latter may itself inhibit iron assimilation.

Protein.

The globulin component of hæmoglobin can be built up from other body proteins, but is, of course, ultimately dependent on adequate intake. Dietary deficiency of protein may therefore be a factor in production of anæmia, particularly in pregnancy; it may also aggravate anæmia already present, or prevent satisfactory utilization of iron given therapeutically. The proteins of most value in promoting hæmoglobin synthesis are those in beef muscle, liver and egg products.

Vitamins.

It has long been known or suspected that many vitamins play a part in erythropoiesis, particularly the *B* complex, which includes folic acid and B_{12}. Nicotinic acid deficiency may be one of the causal factors in the anæmia of pellagra. There is also evidence that a delicate balance between the various components of the *B* complex is essential for normal metabolism, including erythropoiesis, which may therefore be impaired by either excess or deficiency of individual components.

Some dietary sources of folic acid are the green leaves of many plants, notably spinach, as well as mushrooms, liver, kidneys and yeast.

Vitamin B_{12} is now considered, in the light of similarity of natural sources and biological effects, to be closely related to, if not identical with, the "extrinsic" fraction of Castle's hæmopoietic principle ("liver factor"). "Extrinsic" factor is present in beef muscle, milk and eggs and other less common foods, but interaction of the active element in these foods with the "intrinsic" factor present in normal gastric juice is necessary before it can become effective as the hæmopoietic principle. The latter as such, effective without previous interaction with gastric juice, is known to be present in whole liver, brewer's yeast, mammalian kidney and brain.

In addition to its ætiological role in scurvy with its associated anæmia, vitamin *C* appears to have some general stimulant action on hæmopoiesis; as a reducing agent it can convert ferric iron to the more readily absorbable ferrous state. It is abundant in citrus fruit, many berry fruits, tomatoes, swedes, turnips, cauliflower and potatoes. It is readily oxidized and its value reduced or destroyed by long storage or cooking, but not by modern canning processes.

Even this cursory survey makes it evident that while in some countries either poverty or actual shortage of hæmopoietic foods may lead to dietary nutritional anæmia, these factors should rarely operate under existing conditions in Australia. Inadequate intake of hæmopoietic foods does, however, occur, being brought about through ignorance of food values, fads, "slimming", the effort-sparing tea and toast regimen to which persons living alone or harried housewives "too tired to bother about eating" are particularly prone, occasionally by reason of religious tenets, and sometimes in vicious circle with the anorexia or distaste for certain foods resulting from disease. It is not safe to accept perfunctorily a patient's statement that "my diet is all right"; further probing into the actual amount and composition of the meals may reveal surprising inadequacy or imbalance.

Causes Arising Within the Body.

Special Stresses of Physiological Origin.

Special physiological stresses may themselves bring about anæmia-producing conditions, or may accentuate the effect of preexisting potential or actual causal factors.

Growth.

The fœtal hæmopoietic requirements and reserves for the young infant are derived from, and if necessary at the expense of, the mother; unless affected by disease, infants surprisingly are rarely anæmic at birth, even in the presence of quite severe maternal anæmia. In older children and adolescents, dependent on their own labile erythropoietic capacity and reserves, periods of rapid growth may constitute an anæmia-producing stress, especially if complicated by dietary difficulties, or the infections commonly contracted during youth.

Menstruation.

The amount of blood loss regarded as "normal" varies within a wide range. It is likely also that individual women vary in capacity for erythropoietic adjustment to menstrual loss. The normally lower hæmoglobin and red cell levels of women during the reproductive period, as compared with men, and the minor degrees of iron deficiency anæmia not uncommon in the former, are probably related to this periodic stress. On the other hand, in men, even slight anæmia is usually of serious import, and in women, too, it may have pathological rather than, or as well as, physiological significance.

Pregnancy.

The important problem of anæmia in pregnancy has many facets. It has been suggested that pituitary activity may be related to some of the gastric disturbances which occur during pregnancy, which in turn may play a part in production of anæmia, particularly of predominantly macrocytic type. Bolton (1951) suggests that intestinal absorptive dysfunction may also develop, or, if previously present, be accentuated during pregnancy, and that the type of macrocytic anæmia which responds to folic acid (Lawson and Bolton, 1951) is sometimes a nutritional one. The capricious appetite common in pregnancy may also be associated in vicious circle with such gastric and absorptive dysfunction. The demands of the fœtus, particularly for iron, are satisfied at the expense of the maternal reserves, so that if these and the intake of iron are not adequate for compensatory erythropoiesis, the mother will readily develop an iron deficiency anæmia, which may again act in vicious circle with the other ætiological factors predisposing to anæmia during pregnancy.

Disease or Dysfunction.

Diseases in Organs Directly Concerned with Erythropoiesis.

Gastro-Intestinal Tract.—The paramount and varied role played by the gastro-intestinal system in the conversion of dietary "building stones" into the forms in which they can be absorbed and subsequently stored or utilized, is in great part dependent on its delicately balanced enzymes, and, as recent work suggests, on its bacterial flora, which if normally constituted may play a part in the synthesis and possible interaction of folic acid and vitamin B_{12}. Disease in the digestive tract is therefore a frequent and potent cause of anæmia, the type and severity of which will be determined by the predominant defect, or the summation of effects, of several factors. Thus gastric neoplasm may affect chiefly the interaction of extrinsic factor (B_{12}) and intrinsic factor (probably an enzyme), and so cause macrocytic anæmia, with a red cell picture closely resembling that of pernicious anæmia. The picture may, however, be complicated by a nutritional element caused by anorexia, and, if there is ulceration with persistent small hæmorrhages, iron deficiency may also be present. Anæmia in which macrocytosis and hypochromia coexist is often referred to as "dimorphic", an appearance which is very suggestive of gastro-intestinal pathological change.

Ulceration in other parts of the tract, of diverse causation, may similarly give rise to anæmia of varying severity and red cell picture.

Interference with absorption, notably in sprue, both tropical and non-tropical, may cause severe anæmia, either grossly macrocytic, or sometimes dimorphic, or in early or mild cases predominantly hypochromic.

Infective gastro-enteritis may cause anæmia by upsetting the normal enzymal balance, or by changing the bacterial flora either directly or through the action of drugs given for local therapeutic effect, and if accompanied by fever, by "toxic" inhibition of erythropoiesis. Anæmia of mixed ætiology may therefore occur; its quantitative severity may be masked in this, and in other conditions by dehydration or hæmorrhage, if sufficient to cause hæmoconcentration.

Acute infections of short duration, particularly those involving the gastro-intestinal tract, may cause anæmia during convalescence, presumably because only those cells "on the production line" during the illness were affected. In such cases the patient may temporarily present quite severe dimorphic anæmia, which could lead to diagnostic errors.

Liver.—The chief hæmopoietic function of the liver is storage of both iron and hæmopoietic principle. Macrocytic anæmia may occur when the liver is involved in widespread disease.

Bone Marrow.—Primary disease is probably extremely rare, and even "idiopathic" aplastic anæmia may in the future be found to have identifiable ætiology. Efficient performance by the bone marrow is, however, dependent on an adequate supply of iron and hæmopoietic principle, and therefore may be interfered with by nutritional or dyshæmopoietic anæmia.

Some authorities postulate control of bone marrow output by a splenic hormone. The marrow is frequently invaded by neoplastic metastases, leuchæmic infiltration, or abnormalities in its bony covering, all of which may adversely affect its erythropoietic function.

Spleen.—Primary disease of the spleen, as of the bone marrow, is uncommon. Panhæmatocytopenia or erythropenia alone is sometimes apparently due to the primary splenic overaction, but such hypersplenism appears to be more often secondary to irritation or involvement of the spleen in generalized disease processes such as leuchæmia, Hodgkin's disease, tuberculosis, neoplasms and others. The resultant anæmia may be of pure hæmolytic type, but it may include the dyshæmopoietic elements also common in these diseases. Venous back pressure in the portal system, resulting either from thrombosis within the vessels or from external pressure, may cause splenic overaction. Hæmolytic anæmia may occur in association with teratomatous or ovarian cysts, possibly because their abnormal metabolites over-stimulate the spleen. If it is accepted that the spleen controls bone marrow activity, some cases of seemingly aplastic or hypoplastic anæmia could be attributed to splenic overaction.

Endocrine Organs.—There is some evidence suggesting that the pituitary and other endocrine glands exert an influence, as yet ill-defined, on the hæmopoietic system. Anæmia may occur in Simmonds's disease, and is usual in myxœdema; in the latter the anæmia may respond to thyroid therapy alone, or to liver or vitamin B_{12} only if given in conjunction with thyroid.

Menorrhagia in the absence of uterine disease or hæmorrhagic blood dyscrasia is presumably related to some endocrine imbalance, not sufficiently understood to be a certain basis for successful hormonal treatment.

Disease in Organs Not Concerned with Hæmopoiesis.

Disease in organs other than those immediately concerned with hæmopoiesis may cause anæmia indirectly, some instances of which have already been cited. Other examples

are acute or chronic general infections and various toxic states; among the latter are nephritis and other degenerative conditions and the toxæmia which follows severe burns. The anæmia occurring in these circumstances is thought to be generally due to "toxic" inhibition of hæmopoiesis, particularly the utilization of iron. Other generalized diseases, of which *Clostridium welchii* septicæmia, malaria, certain allergies and other conditions such as Lederer's anæmia and acquired hæmolytic anæmia are examples, are characterized by anæmia with hæmolytic features. In some of these, hæmolytic antibodies may be demonstrable by means of the Coombs test and other recently developed techniques.

Any disease process in which hæmorrhage occurs may cause anæmia. External bleeding is, of course, readily recognized, but repeated or chronic small "occult" hæmorrhages, not revealed without special investigations, frequently cause iron-deficiency anæmia, sometimes quite severe. The gastro-intestinal tract is a common site of such hæmorrhages.

Extravasation of blood from the vessels into the tissues, as in severe or repeated purpura or hæmophilia, may in effect resemble external hæmorrhage in giving rise to anæmia, either of orthochromic or in the more chronic cases of hypochromic type.

Constitutional or Hereditary Defects.

Important among constitutional or hereditary defects, and thought to be genetically determined, is the gastric mucosal weakness which leads (usually in middle age) to secretory failure, including that of the "intrinsic factor" and thus to the development of pernicious anæmia. It has been suggested that a somewhat analogous weakness in the intestinal wall is the cause of the absorptive failure associated with sprue.

Of genetic origin are the metabolic or morphological aberrations of Mediterranean anæmia (also known as Cooley's anæmia or thalassæmia), African (sickle-cell) anæmia, and familial hæmolytic icterus. The large flat "target cells" found in Mediterranean anæmia, the bizarre poikilocytes of African anæmia, and the globular spherocytes of familial hæmolytic icterus cannot stand up to the jostling stresses of circulation as well as do the more adaptable disk-shaped normal red cells, and therefore are prone to premature destruction, with resultant anæmia of largely hæmolytic type. Thalassæmia may be a cause of anæmia in a "New Australian" patient of southern European stock.

Blood group incompatibility between mother and child, especially in respect of the Rh factor, though it cannot be regarded as a defect, is a genetically determined cause of *erythroblastosis fœtalis*, in which anæmia is so prominent. Such incompatibility is a chance combination of factors predisposing to an immune reaction which under most circumstances would be biologically protective, but which when operative between mother and fœtus may unfortunately have an adverse effect.

Intake of Harmful Agents.

Intake of harmful chemical or physical agents is being increasingly recognized as an important factor in the production of anæmia, usually of dyshæmopoietic, hypoplastic or aplastic type, but sometimes with features of hæmolysis.

1. Such intake occurs through occupational contact without appropriate precautions. Lead poisoning is a well-known example, but many volatile solvents and other chemicals used in industry have also been incriminated or are suspected. Anæmia may occur at any time while an individual is at risk, but may not be evident for a long time, even years, after the occupational hazard has ceased. In this event, the true cause of the anæmia could easily be overlooked. Hazards of this kind may be present in seemingly innocuous occupations. Dr. D. O. Shiels (1950) reported the case of a typist suffering from anæmia, referred to him because she herself suspected her contact with lead containers of certain duplicating materials. Dr. Shiels's investigations revealed no evidence of lead poisoning, but that she was using benzene to clean her stencils, in a small windowless room, the air in which had a benzol content far beyond the safety limit.

2. The hazards of occupational exposure to X rays or radioactive agents without adequate protection are well known.

3. Accidental intake of substances in general use has occasionally been incriminated as a cause of anæmia. Examples are moth balls (eaten by children), lysol, hair dyes and insecticides.

4. An enormous variety of agents now being used therapeutically, including such universal panaceas as aspirin, phenacetin, and some of the sulphonamides, may, in susceptible individuals, give rise to hæmolytic, hypoplastic or even fatal aplastic anæmia. It is occasionally difficult to determine the relative ætiological responsibility of the disease and the drug being used to treat it.

5. Alcohol, taken in excess, may cause anæmia, probably owing to such effects as disturbance of the balance of the vitamin *B* complex, gastritis, or cirrhosis of the liver.

6. Allergy to certain foodstuffs occasionally results in acute hæmolytic anæmia. The best known example is favism, due to ingestion of the fava bean, used as a food chiefly in southern Europe; as far as is known to the writer, it has not yet migrated to Australia.

Anæmias with No Identifiable Cause.

The already small group of anæmias in which there is no identifiable cause should continue to contract as knowledge increases.

Concluding Remarks.

It has not been possible, within the compass of this article, to do more than outline the subject of the ætiology of anæmia, and to comment briefly on a few salient features. Frequent recourse is acknowledged to the excellent textbooks of Whitby and Britton (1950) and Wintrobe (1951), to which readers are referred for substantiation of many of the statements made above, and for background review and detailed discussion of the knowledge extant at the time of their preparation for publication. Complete survey and critical assessment of more recent knowledge have not been attempted. The great output in recent years of original work and the diversity of journals in which it is published would make the task a formidable one, probably disproportionate to the value as yet assignable to much of this work in relation to its application to the clinical and laboratory investigation of individual cases of anæmia. This large volume of contemporaneous work does, however, emphasize the importance attached to hæmatology in present-day medicine, and the resultant active interest in many of its problems, among which iron metabolism, the relationship between folic acid, vitamin B_{12} and the intestinal flora and their respective roles in hæmopoiesis, and the mechanism of hæmolysis are outstanding.

Lucy M. Bryce,
Melbourne.

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British Medical Association News.

SCIENTIFIC.

A meeting of the New South Wales Branch of the British Medical Association was held at the Rachel Forster Hospital for Women and Children, Redfern, New South Wales, on March 20, 1952. The meeting took the form of a series of clinical demonstrations by members of the honorary medical and surgical staff of the hospital.

Mitral Stenosis with Cerebral Embolus.

Dr. Willa Nelson presented a married woman patient, aged twenty-nine years, who was suffering from a rheumatic heart condition discovered during pregnancy at the age of twenty-one years. She had no history of definite rheumatic fever, but an illness at the age of fourteen years might have been rheumatic fever. Fibrillation had been present for years up to the time of the meeting. The patient had collapsed in July, 1950, with paresis of the right side (face, arm and leg), weakness and loss of speech (aphasia, alexia, astereognosis *et cetera*). Rapid recovery from the hemiparesis had occurred, but speech was slow to recover and

was still defective The patient had symptoms of cardiac failure, but was able to go about quietly.

Examination of the patient revealed a pulse irregular in time and amplitude. The blood pressure was 130 millimetres of mercury (systolic) and 100 millimetres (diastolic). The apex beat was in the sixth left intercostal space five inches from the mid-line and there was cardiac enlargement to the right of the sternum. A diastolic thrill and murmur were detected in the mitral area, with an accentuated second pulmonary sound. No œdema was present. The liver was enlarged one inch below the costal margin. X-ray examination revealed gross enlargement of the heart shadow. The appearances were those of mitral stenosis of long standing. No congestive changes in the lungs were seen. An electrocardiogram showed complete irregularity of rhythm due to auricular fibrillation. Treatment given was digoxin 0·25 milligramme twice a day.

Aplastic Anæmia.

Dr. Nelson's second patient, a married woman, aged sixty-nine years, had a history of severe fatigue and loss of energy for six weeks before her admission to hospital in January, 1952. A blood count at that time revealed a total erythrocyte count of 2,880,000 per cubic millimetre, a hæmoglobin value of 50% (7·7 grammes *per centum*) and a colour index of 0·81. The leucocytes numbered 3750 per cubic millimetre, made up of neutrophile cells 48%, lymphocytes 51%, and mononuclear cells 1%. Platelets numbered 30,000 per cubic millimetre. The anæmia was normocytic and normochromic, with leucopenia and thrombocytopenia. Physical examination revealed, apart from pallor, no positive findings. Dr. Nelson said that three blood transfusions had been given at monthly intervals, but except for improvement temporarily following transfusion, the picture remained essentially that of a normocytic normochromic anæmia, with leucopenia (especially neutropenia) and thrombocytopenia. Occasional macrocytes and microcytes were seen. Marrow examination revealed hypoplastic fatty marrow; cells were scanty and prolonged search was necessary to find 200 cells; the differential count was essentially normal. The result of a Wassermann test was negative. X-ray examination of the skull, shoulder girdle, ribs, pelvis and femur revealed no evidence of multiple myeloma or other lesion. Bence-Jones protein was not present in urine or serum. The gastro-intestinal tract was radiologically normal. Test meal examination revealed normal acidity. No occult blood was found in fæces. Renal function was normal, and the urine was clear. Chest X-ray examination revealed chronic bronchitis and emphysema, with possibly congenital cystic disease.

Discussing differential diagnosis, Dr. Nelson said that there was no evidence of blood loss to suggest anæmia from that source. The findings were against macrocytic and hæmolytic anæmias. Nutritional anæmia was excluded because of no response to a good diet or hæmatinics. Various causes of secondary anæmia could be ruled out: chronic nephritis from the normal renal function and blood pressure; malignant disease from absence of a detected primary lesion; bone disease, poisons, X rays *et cetera* from absence of evidence. The hypoplastic marrow was not against steatorrhœa, but there was no response to treatment with liver. The condition was apparently not leuchæmia, as there was no evidence of primitive cells, and the state of the marrow was not suggestive so far. The provisional diagnosis was refractory anæmia, probably of aplastic type.

Nephritis: Ellis Type 2.

DR. HELEN TAYLOR showed a girl, aged fourteen years, who had been first examined on November 6, 1951, when she had a history of swelling of the legs for six weeks and swelling of the eyes for four weeks. Her general health was good, and there was no history of sore throats. Examination of the patient showed œdema of the legs up to the knees, urine containing one-fifth albumin, and a blood pressure of 158 millimetres of mercury (systolic) and 70 millimetres (diastolic). She was admitted to hospital on the same day and special tests were performed with the following results: plasma protein content total 3·6 grammes *per centum*, albumin 1·4 grammes *per centum*, globulin 2·2 grammes *per centum*; blood cholesterol content 330 milligrammes *per centum*; blood urea content 55 milligrammes *per centum*. Examination of the urinary deposit revealed red blood cells, leucocytes and hyaline and granular casts.

With bed rest and high protein low sodium intake her condition steadily deteriorated. Her weight rose from 119 pounds to 145 pounds on January 14, 1952. In the same period her blood pressure rose to 150 millimetres of mercury (systolic) and 105 millimetres (diastolic). Albuminuria varied from one-quarter to one-third. On January 18 treatment with orally administered cortisone was started. The dosage was 100 milligrammes three times daily for three days, 50 milligrammes twice daily for five days, then 25 milligrammes twice daily for one day. During this treatment her weight increased at a greater rate than before, reaching 155 pounds; the œdema became gross and caused extreme swelling of the vulva; the blood pressure rose to 160 millimetres of mercury (systolic) and 110 millimetres (diastolic); albuminuria varied from one-tenth to one-half. No diuresis occurred after completion of the course of cortisone. On February 2 mercurial diuretic was given intramuscularly with satisfactory diuresis; the injections were continued twice weekly with gradual reduction of œdema and consequent reduction in weight; on February 27 the weight was 136 pounds, the blood pressure 120 millimetres of mercury (systolic) and 80 millimetres (diastolic), albuminuria one-quarter to one-third, and the blood urea content 51 milligrammes *per centum*.

Chronic Non-Leuchæmic Myelosis.

Dr. Taylor's second patient, a married woman, had been first admitted to the Rachel Forster Hospital in March, 1950, complaining that for the past three months she had felt nauseated and food seemed to stick at the lower end of her sternum; she complained also of general malaise. Previous illnesses included jaundice at the age of twelve years. On examination the patient was a healthy looking woman. The liver and the spleen could both be felt two fingers' breadth below the costal margin. A blood count revealed 4,700,000 erythrocytes per cubic millimetre, a hæmoglobin value of 12·8 grammes *per centum* and a colour index of 0·83. The leucocytes totalled 9350 per cubic millimetre. Opaque meal examination showed deformity of the greater curvature of the stomach due to pressure by an enlarged spleen. The response to a Wassermann test of the blood was negative.

On August 11, 1951, the patient was readmitted to hospital. She complained of increased tiredness, breathlessness on effort, nausea, the same feeling of food sticking at the lower end of her sternum, and loss of weight—one stone in two years. On examination of the patient, the liver edge could be felt four fingers' breadth below the costal margin. The spleen was greatly enlarged, extending beyond the umbilicus. No enlargement of peripheral glands was found. A blood count revealed 2,870,000 erythrocytes per cubic millimetre, a hæmoglobin value of 8·7 grammes *per centum* and a colour index of 1·0. Anisocytosis, macrocytosis, microcytosis and poikilocytosis were pronounced. Numerous cells showed diffuse polychromasia. Two erythroblasts and one normoblast were noted. The leucocytes numbered 15,150 per cubic millimetre, being made up of neutrophile cells 79%, lymphocytes 20% and monocytes 1%. Reticulocytes were persistently high in number, averaging 133,440 per cubic millimetre. No increase was found in red cell fragility. Plasma protein values were in normal range and the thymol turbidity test result was negative. The direct Coombs test yielded a negative result.

On September 6 splenectomy was performed by Dr. Kathleen Cuningham through a combined abdomino-thoracic incision. The splenic vein was anastomosed to the renal vein. Severe and pathological bleeding occurred from all wound surfaces and increased towards the end of the operation. The following pathology report was made by Dr. Drury White:

> Spleen weighs 3 lb. 2 oz., and on section is completely homogeneous and the surface is smooth. No obvious fibrosis seen. Colour is moderately dark red. Small splenunculus present, size of a cherry.
>
> There is extreme enlargement of the pulp of the spleen to the exclusion of the sinuses and near exclusion of the lymphoid follicles, though these can still be distinguished and appear normal. The whole of the splenic tissue is homogeneous and extremely cellular. There is practically no fibrosis. The cells consist of all the cells found in the bone marrow; the most being the megakaryocytes and the eosinophil myelocytes. These latter are particularly numerous.
>
> Under examination with the oil immersion lens neutrophil myelocytes, normoblasts, and even erythroblasts can be found. There are also collections of cells with relatively larger nuclei and narrow rims of cytoplasm which are presumably "blast" cells of either the red or white series.
>
> It is evident that the entire spleen has been converted into an extramedullary focus of hæmopoiesis. Hæmosiderosis is absent.

The marrow in this patient, although showing slight increase of erythrogenic activity as compared with leucogenic, was, considering the degree of anæmia, hypoplastic; this fact, with the myeloid metaplasia of the spleen, polychromasia and poikilocytosis of the peripheral blood, anæmia with normal to high white cell count, suggest that the patient may belong to the group of cases designated by British workers chronic non-leukæmic myelosis, or by American workers myeloid-megakaryocytic hepatosplenomegaly.

It would be of interest to know whether there is any X-ray evidence of marrow sclerosis, history of exposure to noxious drugs or irradiations etc.

Dr. Taylor said that after a stormy convalescence the patient had made quite good progress. She had no further dysphagia. The liver had increased enormously in size and filled the whole abdomen. On November 28 blood examination showed a total erythrocyte count of 3,200,000 per cubic millimetre, a hæmoglobin value of 9·8 grammes *per centum* and a total leucocyte count of 41,950 per cubic millimetre. The erythrocyte count and hæmoglobin value had gradually increased and were 4,000,000 per cubic millimetre and 12·0 grammes *per centum* respectively. No X-ray evidence of marrow sclerosis had been found, nor history of exposure to noxious drugs or irradiations. Marrow examination on December 19 yielded the following findings: myeloblasts 1%, myelocytes 3%, metamyelocytes 7%, polymorphonuclear cells 70%, eosinophile cells 4%, lymphocytes 15%. Five erythroblasts and 25 normoblasts were seen in the counting of 200 white cell series. The result of this marrow puncture did not differ essentially from that of August 17. The immature red cells seemed rather more plentiful. The marrow puncture also excluded leuchæmia of the usual type, and it seemed probable that the immature white cells seen in the peripheral blood were coming from the extramedullary centres of hæmatopoiesis.

Retrosternal Goitre.

DR. NAOMI WING presented a woman patient, aged sixty years, who had attended the out-patient department in October, 1951, complaining of severe breathlessness, palpitation and precordial pain, accompanied by cough and frequent small hæmoptyses. She had also weakness of the legs and pronounced ataxia. Twenty years previously she had consulted Dr. Hugh Poate, who had diagnosed acute thyrotoxicosis with cardiac failure. She was treated for many years by Dr. E. H. Stokes at the out-patient department at Sydney Hospital, where she had the treatment which was then adopted, namely, Lugol's iodine. She had gradually improved; the swelling that had been present in her neck diminished, and she was able to carry on her work. Intervals of bed rest and digitalis therapy were required over the years. Later she had complained of numbness and tingling in the legs and feet, with great difficulty in walking, sore tongue and loss of appetite. She was treated at Saint Vincent's Hospital out-patient department with "Campolon" and vitamin B_1 injections with beneficial results. She returned to work and discontinued treatment, but became ill again, collapsed and was admitted to Saint Vincent's Hospital where extensive investigations were carried out in June, 1951.

On examination of the patient at the Rachel Forster Hospital on October 24 her weight was six stone three pounds. She was a thin, pallid, poorly nourished woman, with moist skin, hands perspiring freely and pronounced tremor. Examination of the tongue showed pronounced glossitis, and there was cheilosis at the corners of the mouth. The blood pressure was 200 millimetres of mercury (systolic) and 100 millimetres (diastolic). The pulse rate was 150 per minute, and there were some extrasystoles. Slightly increased venous pulsation was visible in the neck. The apex beat was in the sixth left intercostal space three and a half inches from the mid-line. A rough systolic murmur was audible at the aortic area. Slight enlargement of the thyroid was visible in the neck. Dulness was detected on percussion over the upper part of the sternum and the trachea was displaced to the right side. The breath sounds were poor at both lung apices, but clear elsewhere; no moist signs were present. The abdomen was soft and slightly tender below the right costal margin. The liver was not palpable. Severe pain and tenderness were present over the lower lumbar part of the spine and sacrum. The pupils were equal and reacted to light and accommodation. The cranial nerves were intact. No weakness of muscles was found in the upper extremities. The lower extremities were both very weak. Rombergism was present, with tingling in both feet. The plantar reflex was flexor on both sides. X-ray examination of the lumbar spine, pelvis and left hip joint revealed no bony abnormality in the lumbar spine, apart from minimal arthritic lipping and incomplete sacralization of the first sacral vertebra of congenital origin. There was also pronounced angulation of the middle segment of the sacrum, the lower segment being bent forward at a right angle. No other abnormality was seen in the pelvis or hip. The lung fields were clear radiologically, but there was widening of the upper part of the mediastinum with deviation of the trachea to the right, possibly due to a retrosternal thyroid. Subsequent lateral X-ray examination of the chest revealed no direct evidence of intrathoracic thyroid. A blood count in October, 1951, showed 4,720,000 erythrocytes per cubic millimetre, a hæmoglobin value of 14·3 grammes *per centum* (96%) and a colour index of 1·02. The leucocytes totalled 9800 per cubic millimetre, made up of neutrophile cells 69%, lymphocytes 28%, monocytes 1%, eosinophile cells 1% and basophile cells 1%. The blood sedimentation rate was 29 millimetres (normal 0 to 20 millimetres). On March 10, 1952, the basal metabolic rate was −1% and the weight was six stone three pounds.

A full report was obtained of tests carried out at Saint Vincent's Hospital in June, 1951. The marrow smear examination report stated that in the granular series there was a slight shift in the direction of maturity. The ratio of white to red cell series was 3:1. The films showed no significant abnormality. Liver biopsy revealed no significant change, and liver function tests displayed no abnormality. A blood count showed 4,610,000 erythrocytes per cubic millimetre, a hæmoglobin value of 86% (11·9 grammes *per centum*), and a colour index of 0·87. Pallor and polychromasia were slight. The leucocytes numbered 4800 per cubic millimetre.

Treatment was begun on October 24, 1951, with vitamin B_{12}, 20 milligrammes three times a week, for changes in the central nervous system in view of the long history of treatment for macrocytic anæmia, with good effect. When the injections were discontinued, numbness, tingling and ataxia returned. On December 6 "Ferrivennin", five injections each of five millilitres, was given. The colour remained very poor and the general condition showed no improvement. On December 12 methyl thiouracil, 0·05 gramme three times a day, was commenced and given for two weeks. The pulse rate came down to 80 per minute, there was less tremor, and the patient felt much better. Then severe attacks of breathlessness occurred, and administration of thiouracil was discontinued on January 30, 1952. The pulse rate steadily increased, and breathlessness and slight hæmoptyses continued. On March 12 methyl thiouracil therapy was recommenced, and by March 19 the patient was feeling better again. When called in consultation, a surgeon did not consider surgery justifiable.

Hypertension Treated with Hexamethonium Bromide.

DR. JEAN PALMER showed a married woman, aged sixty-two years, suffering from hypertension first diagnosed fourteen years previously. She had had headaches and dizziness for twelve years and "blackouts" for seven years. The attacks were preceded by blurring of vision and severe headaches, and there was temporary loss of consciousness. The patient had had sudden hemiparesis with dysarthria three years before the time of the meeting, followed by complete recovery. She had experienced excessive fatigue, exertional dyspnœa and palpitations for two years. On examination she was found to have thickening of peripheral vessels. The pulse was regular. The apex beat was displaced to the left half an inch outside the mid-clavicular line in the fifth left intercostal space. Presystolic gallop rhythm was present, with an accentuated aortic second sound and a grade I systolic murmur maximal at the apex. The blood pressure was 260 millimetres of mercury (systolic) and 150 millimetres (diastolic). The fundi showed grade II hypertensive changes with arteriolar narrowing and tortuosity, arterio-venous nipping and congestion of veins.

After one week's bed rest the blood pressure fell to 230 millimetres of mercury (systolic) and 110 millimetres (diastolic). Hexamethonium bromide therapy was commenced, with an initial dose of one tablet (250 milligrammes) thrice daily. The dose was increased by one-half tablet daily to a total dose of four grammes per day. The blood pressure on the patient's discharge from hospital in the fifth week was 160 millimetres of mercury (systolic) and 90 millimetres (diastolic). Oral maintenance treatment had been continued for the past six weeks, the present dose being two grammes per day. Weekly blood pressure readings had varied from 130 millimetres of mercury (systolic) and 80 millimetres (diastolic) to 160 millimetres of mercury (systolic) and 90 millimetres (diastolic). Six weeks from the commencement of treatment bromide intoxication became apparent and the bitartrate salt of hexamethonium was

substituted for the bromide with great improvement. The patient was now free of headaches and dizziness, and her exercise tolerance was much increased. She was taking digoxin tablets, one twice a day, in addition to the hexamethonium. Salt restriction had recently been relaxed with no apparent ill-effect.

Familial Depression of the Sternum.

Dr. Palmer's second patient, a married woman, aged thirty-eight years, had as her only complaints pain in the left side of the chest extending from the infrascapular area to the precordium, mild exertional dyspnœa and winter bronchitis for several years. The chest pain had diminished since she had left heavy cleaning work. She had had pneumonia at the age of twenty-three years, for which X-ray examination was made. Cardiac enlargement was diagnosed at that time. Investigation of the family history revealed that the patient's mother had a congenital sternal deformity and bronchial asthma; she had died at the age of forty-seven years. One brother had a "pigeon chest" deformity and one brother had asthma. There were two normal siblings. Examination of the lower half of the sternum showed a saucer-shaped depression with a deep groove running horizontally. On palpation the whole precordium was quiet and the apex beat was not palpable. The first mitral and second pulmonary sounds were split. A grade II systolic murmur was audible down the left border of the sternum. Venous pressure was normal. No clubbing of the fingers or cyanosis was present. Breath sounds were normal. The blood pressure was within normal range. X-ray examination of the chest showed a great increase in the width of the cardiac shadow both to the right and to the left. The heart was abnormally translucent owing to the flattening. In the oblique views the distension caused both ventricles to appear enlarged. In the barium-filled œsophagus two unusually distinct indentations due to the aorta and pulmonary artery were seen. The lung fields were normal. In the electrocardiogram, the *P* waves were 2·5 millimetres in height in lead II, appearing tall and broad in comparison with the low voltage *QRS* complexes. There was notching of the *QRS* complexes, with delayed intraventricular conduction. No evidence of right ventricular hypertrophy was present.

Chronic Vitamin B Deficiency.

Dr. Palmer's third patient, a married woman, aged forty years, had been first examined in June, 1951, when she complained of pain in the calves and swelling of the legs for two and a half years, weakness of the legs and numbness of the feet with occasional falling, dyspnœa and palpitations on exertion, excessive sweating and redness of the hands and feet, increase of three stone in weight and oliguria, and anorexia. She had been previously employed as a barmaid and had had a steady alcoholic intake, mostly wine, over some years. Her diet was poor owing to anorexia. She had been previously treated with bed rest and mercurial diuretics with temporary reduction of œdema.

On examination, the patient was found to be a plethoric woman with blotchy bluish-red discoloration and swelling of the fingers and toes. She was sweating excessively, and there was gross œdema of the legs to the lower third of the thigh. Muscular weakness and impaired sensation of the lower limbs were present, with tenderness of the calves. Knee and ankle jerks were absent. The pulse rate was 100 per minute. The heart was enlarged, with displacement of the apex beat to one inch outside the mid-clavicular line. A grade II systolic murmur was audible, being maximal in the third left intercostal space close to the sternum. The blood pressure was 180 millimetres of mercury (systolic) and 90 millimetres (diastolic). Moist râles were heard at both lung bases. X-ray examination with fluoroscopy showed generalized cardiac enlargement; the cardio-thoracic ratio was 1·7, and congestive changes were seen in both lung fields. An electrocardiogram showed a vertical heart with no abnormality except possibly the inverted *T* wave in lead III. The basal metabolic rate was −1%. The urine was normal and the blood urea content was 41 milligrammes *per centum*. Urea concentration was impaired. Leucocytes numbered 21,000 per cubic millimetre with a normal differential count.

The patient was given vitamin *B* therapy, 50 milligrammes three times daily, and this was continued with gradually diminishing doses for the next six months. No therapy had been given for the last two months. The œdema had disappeared within two weeks of commencement of treatment and had not recurred. Muscle power had improved more gradually. The knee jerks were still absent, and there was still calf tenderness after six weeks. Recent examination had shown normal knee jerks; ankle jerks were present but diminished. The heart size had decreased rapidly at first, and the systolic murmur had disappeared; further diminution in size was apparent in recent X-ray pictures. The hypertension had persisted, as had the impaired renal function. The patient probably had chronic nephritis in addition to the nutritional deficiency. The electrocardiogram had shown many changes, including the *T* wave's becoming upright in *AVL* and lead III.

(To be continued.)

Out of the Past.

In this column will be published from time to time extracts, taken from medical journals, newspapers, official and historical records, diaries and so on, dealing with events connected with the early medical history of Australia.

THE QUARANTINE REGULATIONS.[1]

[*Sydney Gazette*, Thursday, February 1, 1838.]

By recent intelligence from Spring Cove we learn that up to Monday last the Scotch Immigrants by the *Minerva* now suffering under the ravages of typhus fever, had not been landed although we are told that imperative orders to that effect had been forwarded from the Colonial Secretarys Office on the Wednesday previous. What the reasons are which have induced the Surgeon in charge to delay releasing the sick, as well as the healthy from the confined, and in this case necessarily polluted air of a ship, to the pure air, and refreshing breezes of the land, it is difficult for us to determine. Another passenger, a young and interesting female, fell a victim to the ravages of the disease on Saturday, leaving a helpless infant to suffer the loss of a mother's care. On Monday, it was understood, the Immigrants would be landed, and placed in the houses recently erected at the Quarantine Station for that purpose.

It is difficult to conceive why the Government should wish to conceal the actual state of the disease in vessels under Quarantine, as they have invariably done in the cases of the *Canton*, the *Lady McNaughten*, the *John Barry* and the *Minerva*: or if it is not their wish to conceal it, why do not they direct, for general information, the publication of the state of the hospital, and the names of those who fall victims to the ravages of the disease. We have reason to believe that no objection to that course exists in the medical department: it is with the Executive Government alone, therefore, that the blame rests. Much anxiety is felt by the public generally, to know the progress which the fever is making, as well as by individuals who have relatives or intimate acquaintances on board, to ascertain whether they are yet in the land of the living.

It would require no great exertion on the part of the Government to put them daily in possession of the desired information: and we are sure were the Acting Governor to follow the dictates of his own heart, the information would not long be withheld. How different has been the conduct of the Van Diemen's Land Executive in the case of the ship *Bussorah Merchant* just arrived at Hobart Town with Irish Emigrants from Cork, placed in quarantine in consequence of the existence of typhus fever and a variety of other diseases on board? As soon as it was practicable after her arrival, the Colonial Secretary, by direction of the Governor Sir John Franklin, published for general information in the *Government Gazette* of the sister colony, a complete list, not of the sick and the dead merely, but of all the Emigrants who left Cork in the vessel, the place from which each passenger emigrated, his age, trade, and number of his family, the names of the deaths and the disease of which each individual died. To the New South Wales Government we can only say—go and do likewise.

[1] From the original in the Mitchell Library, Sydney.

Correspondence.

DYSFUNCTION.

SIR: At one meeting during the last week, in the discussion on a case of not fully clarified thyrotoxicosis, one of the doctors commented: "It is very likely a case of hyperfunction of the thyroid gland, a dysfunction of it." What is the meaning of dysfunction? Did the phrase hit the nail

on the head? To my mind it did not, not at all.. It is not an exact definition of a well-defined condition. Its meaning is just this: "The function is bad, but I do not know anything more about it." In "Blakiston's New Gould Medical Dictionary" we can read: "Dysfunction. Any abnormality or impairment of function, as of an organ."

Let us make a short analysis of dysfunction. Dys- is a Greek prefix. "In medicine denoting *difficult* or *painful*; *faulty* or *impaired*; or *abnormal* or *morbid*" ("Blakiston's New Gould Medical Dictionary"). It is therefore the opposite of eu-. In Liddell and Scott's "Greek-English Lexicon" we read: "*δυσ-*, prefix, opp. to *εὖ-* . . . *un-* or *mis-* . . . with notion of *hard, bad, unlucky.*" In French it is *trouble fonctionnelle,* in German *Funktionstöhrung* (Veillon, 1951). It is frequently used in the medical literature. In the above-mentioned dictionary there are 113 words beginning with dys-, starting with dysacousia and ending with dysuria. Dyspnœa is "difficult or labored breathing". Dysmenorrhœa is "difficult or painful menstruation". Dyspepsia is "disturbed digestion". Dysendocrinism is "any abnormality in the function of the endocrine glands". Are hyperfunction and hypofunction within the field of dysfunction? The answer is in the affirmative, if either of them produces symptoms of a disease. I would go even further. Dysfunction for the time being means either hyperfunction or hypofunction. The term dysfunction is used in this sense in Kolmer's book "Clinical Diagnosis" (Kolmer, 1949). We read under the heading "Endocrine Dysfunction" the following lines: "The glucose tolerance test is used chiefly for the detection of impairment of carbohydrate metabolism in diabetes mellitus due to a deficiency in the production of insulin by the pancreas. But other *endocrine dysfunctions* may likewise affect carbohydrate metabolism and the results of glucose tolerance tests. For example, an exaggerated response due to diminished glucose tolerance may occur in *hyperpituitarism* similar to that observed in mild diabetes while *hypothyroidism* and *hypoadrenalism* may show just the reverse or a decreased response due to increased glucose tolerance." (All italics are mine.)

But dealing with endocrine dysfunctions I have to point out that just in endocrinology the exact use of the term dysfunction is of paramount importance. This is the main reason for my letter. In this field some quite outstanding authors use the word dysfunction in another sense than usually denoting with it the effect of a supposed "abnormal" hormone. They also use it in the same sense as Plummer used the word dysthyroidismus so many years ago. Plummer suggested that in dysthyroidismus the gland formed not only an excess of the hormone but also one which was abnormal as a result of incomplete saturation of its molecule with iodine (quoted in Best and Taylor, 1945). In Zondek's book (Zondek, 1935) we read similar thoughts: "The question has been much debated whether the disease [Graves's disease] is due to pure hyperthyroidism or *dysthyroidism,* i.e. whether the secretion of the gland is abnormal in quantity or quality. It is very difficult to decide this point with certainty." Reifenstein (Sodeman, 1950) writes: "It is beyond the scope of this chapter to discuss in detail the pathology of the various disturbances that are recognized as endocrine dysfunctions. These disturbances may be divided roughly into (1) hyperfunction, (2) hypofunction and (3) dysfunction." (See also about "abnormal" hormones on page 515 of the same book.)

Hormones are produced in the organism by a very long and complicated chain of chemical reactions. It is not only possible, it is very likely or even certain that these reactions may stop at any time at any stage earlier or might proceed over the borderline, so that production of "abnormal" hormones is very probable. But at least heretofore there are no clinical pictures, syndromes or diseases known which are to be ascribed with certainty to the effect of abnormal hormones. There is at the time being no more than a simple guessing about the action of these supposed hormones. In contrast to the above-quoted authors there are a lot of books of internal medicine, pathology and endocrinology which do not even mention the possibility of such conditions. In the book of Means (1948) and in that of Rundle (1951) on the diseases of the thyroid gland the term dysthyroidismus is not used. And I would quote Selye (1950), in whose quite recent book we can read: "Signs of hyper- and hypothyroidism may occur simultaneously in the same patient: Exophthalmos + diminished thyroid function. Such complications have *erroneously* been ascribed to the production of an abnormal thyroid hormone derivative and were described as *'dysthyroidism'*; they are now more commonly attributed to increased thyrotrophin secretion." (Italics are mine.)

I think for the time being it is not justified and not correct to speak and write about "dysfunction" in the sense of a disease caused by an abnormal hormone. If in the future the existence of such condition should be proved, then in my opinion the condition has to be denominated by a newly created expression, the meaning of which would be distinct and precise and not so problematic and ambiguous as "dysfunction".

I believe at present we should use "dysfunction" merely in the sense of wrong function; it should not mean more. But if so then we should not say, "It is a hyperfunction, or rather a dysfunction", but instead say, "It is a dysfunction of an organ, or rather a hyperfunction". Or in the opposite condition, "It is a dysfunction, or rather a hypofunction".

Yours, etc.,

22 Ocean Avenue,
Edgecliff,
New South Wales.
June 3, 1952.

CHARLES ENGEL.

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THE MEDICAL BENEVOLENT ASSOCIATION OF NEW SOUTH WALES.

SIR: This society has just published a very remarkable document, namely, its annual statement of accounts. Therein is revealed the fact that there are less than 750 practising doctors in New South Wales who can find time and thought to contribute the minute sum of fivepence per week for the relief of those who need the assistance of this Association. Surely every practitioner who reads this will recognize his duty immediately to enrol as a member and pay at least the subscription of a guinea per year.

Yours, etc.,

193 Macquarie Street,
Sydney,
June 18, 1952.

ERIC P. BLASHKI.

SUB-OXYGENATION AND ANÆSTHESIA FOR TONSILLECTOMY.

SIR: The development of the para-magnetic oxygen analyser, and more recently of complex photoelectric equipment capable of determining the oxygen saturation of hæmoglobin in circulating blood, has shown much regarding the oxygenation of the anæsthetized patient. With regard to ether and air anæsthesia, it has been shown (as would be expected from physical considerations) that the oxygen concentration in the inspired vapours is somewhat below atmospheric, the degree depending on the ether vapour content and the degree of rebreathing of expired gases. Also with an ether and air anæsthetic the arterial oxygenation saturation of hæmoglobin is below the normal 95%, though the patient's colour is not changed to the human eye.

The almost universal practice of adding a little oxygen to the ether-air mixture corrects this minor degree of sub-oxygenation. However, in the administration of tonsil anæsthetics the ether is usually vaporized with air alone; presumably these patients are mildly sub-oxygenated even when the anæsthetic is perfectly given. Difficulties with the anæsthetic, such as coughing, crowing, or breath-holding, rapidly produce cyanosis, since the initial sub-oxygenation implies a narrowed margin before the onset of cyanosis.

This sub-oxygenation is avoided readily by fitting a "T" or "Y" piece to the inlet of the vaporizer and feeding air through one limb and oxygen at about two litres per minute through the other. If difficulties are encountered with the anæsthetic the oxygen flow can be increased as necessary. When "Trilene" is used instead of ether, the addition of

oxygen is again advantageous, although because of the much lower percentage concentrations of the "Trilene" vapour used, the necessity of the added oxygen is probably less.

When the source of compressed air is an electrically driven pump, mounted near the ether vaporizer, it is undesirable to add oxygen as this addition creates a violently explosive mixture. It is probably better to have a mildly sub-oxygenated patient than run the risk of having one splattered over the walls of the operating theatre.

Yours, etc.,

Melbourne, June 13, 1952. W. H. J. COLE.

NATIONAL HEALTH SCHEMES.

SIR: There is a dangerous aspect of the so-called Earle Page health scheme which is not being given enough attention by the profession and politicians at large, and that is, how the public are going to be coerced into joining the scheme. It is obvious that solvency will depend on there being enough contributors who are up to date with their payments. In Britain, where there have been many "medical schemes", it was found that everywhere the people who used the schemes most were the most irregular in their payments.

Furthermore, it was found that no scheme was successful unless there was some kind of pseudo-compulsory method of collection, such as deduction from wages at a works office and so on, until the notorious National Health Insurance or panel scheme with its national compulsory deduction from all workers below a certain wage level was reached. This existed from 1911 approximately until the present British scheme came into existence some years ago and which covered everybody from national income.

But the panel scheme was a friendly society organization similar to the Earle Page arrangement. It was built up on a system of spying on the patients with "health visitors" and grinding down the doctors with decreasing *per capita* payments, and surcharges for "overprescribing" with such intensity that it earned from a judge of the High Courts, in a book he wrote, the title of "The New Despotism".

Of course, Australia is not heading for such a scheme. In that case Sir Earle Page will be able to say with ease: (*a*) how people are going to be made join up, and (*b*) how payments are to be kept up to date.

Can he do so? Can he say, there is not a powerful wedge being forged, of which the Pensioners Medical Scheme is the thin edge?

If not, we can look forward to some form of compulsion, with control of the doctors at large organized successfully by the vested interests giving, as actually occurred in Britain, many British Medical Association officials well-paid part-time jobs as medical examiners and so on.

Yours, etc.,

Cygnet, Tasmania, June 10, 1952. JOHN A. MCCLUSKIE.

GENERAL PRACTITIONER GROUPS.

SIR: The General Practitioner Group in Queensland is still active and busy, looking after the welfare of general practitioners throughout the State, and our Branch Council is fully cooperative, in that it refers all matters concerning general practitioners to that committee for its consideration and recommendations.

This appears to be a very wise plan, for the general practitioners form the backbone of the profession, and by far the largest percentage of practitioners in every community, and such general practitioner groups should be active and progressive in every State; many years of experience have taught us that the general run of members of the British Medical Association take little active interest in its affairs, unless their individual interests, particularly their pockets, are threatened.

Do these general practitioner groups still exist in other States? If not they should do so, and I would suggest that a Federal Council representative of each State should be one of the members of its committee. Further, I would suggest that a central (or Federal) committee of these general practitioner groups should be formed, consisting of one member from each State to consider its problems and determine its policy throughout the Commonwealth for the guidance of the Federal Council itself in general practitioner matters.

In times of peace prepare for war.

Today the situation seems fraught with possibilities of government interference in various directions. The problem of hospital treatment is a very real one; in Queensland there is no means test, and country hospital medical officers, with right of private practice, are feeling the pinch. Further, we have committed ourselves to a Commonwealth medical scheme for the care of pensioners, which includes widows and invalids as well as old-age participants. We have also accepted a pharmaceutical benefits scheme open to all and a repatriation scheme. All are now in full swing, and it behoves us to see that there is an abuse of none.

Suppose we had a change of government and the means test was abolished all round; where would the medical profession be, unless we are awake and active and properly organized? Are general practitioners awake to these possibilities, or are they going to remain uninterested and asleep till the threat is at their very door? Cannot the nation's experience in World Wars I and II stand as a warning to us? In Queensland we have a full representation on the Branch Council, and, as I have said before, the General Practitioner Committee has afforded quite considerable help to that body in dealing with questions that concern general practice. As all know, social conditions and circumstances that concern all types of medical practice are changing so rapidly today that it is imperative that medical men generally be on the alert, and actively interested and organized, so that they may present a ready and united answer to any problem that presents.

I am sure that the secretary of the General Practitioner Group in Queensland will be very happy to answer questions that may be put to him concerning this organization here.

I repeat the saying—in times of peace prepare for war.

Yours, etc.,

1 Burch Road, Annerley, Brisbane. June 17, 1952. L. P. WINTERBOTHAM

CHILDREN AND ACCIDENTAL POISONING.

SIR: Dr. D. J. Brennan, in his recent letter (June 7, 1952), issued a timely warning against the danger of accidental poisoning in children, in particular stressing the danger from some of the more familiar home remedies, such as "Benzedrine" and gentian violet capsules, for precautions taken against accidental poisoning by such seemingly innocuous substances are not as careful as those taken against more obviously poisonous substances. The cases he quoted recall to mind an experience while senior resident at the Marrickville District Hospital.

Late one Friday afternoon I was urgently summoned to the casualty department, there to find a distraught mother with her happy, cheerful, three-year-old boy, probably wondering what all the excitement was about and whether it really concerned him at all. It appeared that more than an hour before he had swallowed about seventy 0·25 milligramme digoxin tablets. He had been visiting his grandmother, who had commenced digoxin treatment on that day, having had a prescription for 100 digoxin tablets dispensed by her chemist that morning. Finding his mother and grandmother deeply engrossed in conversation, the youngster strolled into the bedroom, took the bottle from the bedside table, and swallowed about 70 tablets before being discovered. The delay in coming to hospital was caused by first consulting the chemist. The child was given a stomach wash-out and admitted to hospital. In so far as could be judged from the residue obtained, a large proportion of the ingested digoxin had already been absorbed by the stomach mucosa. At this stage his pulse and heart sounds were normal in every respect. A search in all the text-books then available, including large works on medicine and toxicology, did not indicate what further measures to adopt in such a case, other than the standard advice to suspend the exhibition of any of the digitalis group of drugs temporarily if one or more of the toxic effects due to overdosage should occur. Some two hours later, the child's pulse rate suddenly dropped, and the pulse became completely irregular in time and amplitude, the rate within seconds varying from 60 to 100 per minute, and at times the heart ceased beating altogether for several seconds at a time. The child was now desperately ill, with the constant danger of ventricular fibrillation supervening, these cardiac irregularities and his critical condition lasting more than two days. The two drugs employed in counteracting these effects to some degree were comparatively large doses of atropine by subcutaneous injection and oral quinidine. Other than that there appears to be no actual antidote to a gross overdose of one of the

digitalis group of drugs. From the purely academic point of view it is regrettable that, because of an acute abdominal operation at the same time, a more or less continuous electrocardiogram could not then be taken. The child subsequently made a complete recovery, with no cardiac abnormality detected.

Time and again, in hospital practice, one notes with surprise how many children will consume large numbers of tablets, which many adults will swallow only with difficulty. This should call for greater care in keeping even non-dangerous tablets out of the reach of children in order to avoid even minor upsets. To illustrate this point further I recall an amusing incident in my early childhood when a playmate of mine, more enterprising and less discerning in matters gastronomic than I, at one sitting ate a cake of soap and drank the contents of a full large-sized bottle of ink, seemingly with much relish. Let the full story be known. To my undying shame, I reported the matter to his mother, although the remains of his repast were clearly on his face for anyone to see. However, other than some slight punishment for his misdemeanour, he suffered no ill effects after his experience.

Patients are usually requested on prescription forms to bring the prescription along on their next visit. Some practitioners have also adopted the practice of adding some sentence such as "Keep all medicines out of toddlers' reach" or similar warning. This practice has much to commend it, and may result in that small degree of extra care which will make impossible cases such as that quoted, which might quite easily have ended as a family tragedy.

Yours, etc.,

J. SCHNEEWEISS.

30 Penkivil Street,
Bondi,
New South Wales.

June 18, 1952.

SUCCINYLCHOLINE CHLORIDE: AN ULTRA-SHORT-ACTING RELAXANT.

SIR: We would like to thank Dr. Stace for his congratulatory remarks on our article of April 19 in THE MEDICAL JOURNAL OF AUSTRALIA. In particular we strongly endorse his mention of the use of "Scoline" for facilitation of œsophagoscopy and bronchoscopy. Several ear, nose and throat surgeons have remarked, and it is also our personal observation, that it is the only agent to date that produces complete immobility of the vocal chords in the abducted position. Dr. Stace disagrees with us on one point only—the amount of thiopentone to be used with "Scoline". It has been our continued experience that, given a similar type and weight of patient, we can achieve equal or greater ease of intubation with roughly half the amount of thiopentone to that used with C10. By employing minimum doses of "Pentothal" we were endeavouring, in our preliminary trials, to assess the real value of "Scoline" as a relaxing agent facilitating intubation, as it is known that some anæsthetists use three-quarters of a gramme of thiopentone as a sole agent for intubation.

This month's excellent articles of Dr. Orton and Dr. Egerton Williams, coming as they do from another State, offer further proof of the efficacy of "Scoline". In particular we were pleased to note that Dr. Williams endorses our views that thiopentone and "Scoline" can be mixed in the same syringe subject to certain precautions being observed. In this respect he disagrees with his colleague Dr. Orton. However, we are sure that the "Scoline" in such a mixture retains its potency for up to ten minutes. Further we have demonstrated to the New South Wales Section of the Australian Society of Anæsthetists on March 18 this year that only a two to one ratio of thiopentone to "Scoline" is required to ensure miscibility. As regards the release of histamine, all our skin test doses with "Scoline" produced flare which may be related to its low pH.

No evidence of bronchospasm or laryngospasm has presented itself.

We would like to take this opportunity to record that "Scoline" exhibits its normal relaxation and ephemeral action when used to reinforce C10. "Scoline" is entirely satisfactory for children, and we have used it down to the age of four years.

Yours, etc.,

A. P. BALTHASAR.
C. A. SARA.

193 Macquarie Street,
Sydney,

June 19, 1952.

Medical Practice.

ENCEPHALITIS IN SYDNEY: AN EXPLOSIVE OUTBREAK ASSOCIATED WITH A HITHERTO UNDESCRIBED VIRUS.

THE following statement is submitted by Mr. N. F. Stanley, B.Sc., Acting Director, Institute of Epidemiology and Preventive Medicine at the Prince Henry Hospital, Sydney, in order to draw attention to a virus disease likely to be prevalent in summer months, and to the importance of the laboratory diagnosis of infections with "neurotropic" viruses.

In the course of epidemiological studies with poliomyelitis and Coxsackie viruses in Australia, it became evident that in January of this year a dramatic increase had occurred in the number of cases of encephalitis admitted to hospital. After four weeks there was a rapid decline in incidence. It is the purpose of this preliminary communication to note very briefly the main clinical features observed by medical officers in charge of the patients and reported to me and to describe some of the properties of a Coxsackie virus believed to be the agent responsible for the explosive outbreak.

There were no fatal cases. With few exceptions the patients made an uneventful recovery. The findings in order of frequency were pyrexia, headache, stiff neck, lassitude, pleocytosis in the cerebro-spinal fluid, vomiting and dizziness. The majority of patients were females in the limited age group of seventeen to twenty-three years. The distribution of cases appeared to be fairly even over the Sydney metropolitan area. A final diagnosis of encephalitis was made in each instance without knowledge of the findings of the virus diagnostic laboratory. The disease is in no way to be related to Murray Valley encephalitis.

In thirty cases examinations were made for the presence of poliomyelitis, encephalitis and Coxsackie viruses. Ten strains of an infective agent were isolated from the fæces of patients during the acute phase of the disease. The infective agent was shown to be pathogenic only for suckling mice twenty-four hours old and has been identified as a Coxsackie virus. Because the strains possess properties quite distinct from Coxsackie viruses of group A or group B, they have been provisionally designated group C. All strains isolated during the outbreak were serologically identical. They produce an encephalitis following the intraperitoneal inoculation of suckling mice.

No lesions in the skeletal muscle or pancreas have been observed. The incubation period may be from five to fifteen days, when mice become emaciated and develop wrist drop and tremors, and die slowly. Mice older than twenty-four hours are almost insusceptible to infection. The virus may be isolated in high concentration from the brains of infected mice, but is difficult to demonstrate in skeletal muscle. It is serologically distinct from other Coxsackie viruses isolated in this country.

The antibody response of patients from whom virus was isolated and from others with the same clinical picture, but from whom no virus was isolated, was high. Neutralizing antibodies were often detected in convalescent sera at dilutions of > 1 in 1280. The sera collected during January from patients not showing symptoms of encephalitis have so far shown no evidence of neutralizing antibodies at a serum dilution of 1 in 20.

Attention has been drawn to a mild but explosive outbreak of encephalitis associated with a Coxsackie virus. The virus diagnostic laboratory of the Institute of Epidemiology and Preventive Medicine at the Prince Henry Hospital is now in a position to carry out extensive serological investigations on patients showing a similar clinical picture in an attempt to gain further epidemiological data on Coxsackie infections in Australia.

University Intelligence.

UNIVERSITY OF SYDNEY.

PROFESSOR LORIMER F. DODS has been granted leave of absence from April to November, 1952, to enable him to take up a National Health and Medical Research Council scholarship and post-graduate studies overseas.

Dr. K. W. Cleland has been granted an additional year's leave of absence without pay in order to continue his work at Cambridge.

Dr. B. L. Reid, a graduate of the Faculty of Medicine and the Faculty of Veterinary Science of the University of Sydney, has been appointed to the lectureship in histology and embryology.

Post-Graduate Work.

THE POST-GRADUATE COMMITTEE IN MEDICINE IN THE UNIVERSITY OF SYDNEY.

Hallstrom Institute of Cardiology, Royal Prince Alfred Hospital.

THE second annual post-graduate course, conducted by the Hallstrom Institute of Cardiology, in conjunction with the Post-Graduate Committee in Medicine, will be held in the Scot Skirving Lecture Theatre, Royal Prince Alfred Hospital, from July 14 to 25, 1952. The course will consist of a series of ten afternoon lectures followed by demonstrations of patients and other relevant material. Emphasis will be placed this year on the modern theory, practice and interpretation of electrocardiograms, and this section of the course will be open to electrocardiograph technicians employed by hospitals and the armed services. The supervisor will be Dr. J. F. Farrar.

Programme.

The programme will be as follows:

Monday, July 14: 2 p.m., registration, introduction and lecture, "Physical Basis of Electrocardiography I. The Normal Electrocardiograph", Dr. Bruce Sinclair-Smith; 3.30 p.m., demonstration of electrocardiographic apparatus, Mr. W. Bell.

Tuesday, July 15: 2 p.m., "Physical Basis of Electrocardiography II. Ventricular Hypertrophy. Unipolar Electrocardiography (Vector Cardiography)", Dr. Bruce Sinclair-Smith; 3.30 p.m., clinic, Dr. J. K. Maddox and Dr. Sinclair-Smith.

Wednesday, July 16: 2 p.m., "Electrocardiography of the Arrhythmias I", Dr. J. Halliday; 3.30 p.m., ward round, Dr. J. K. Maddox.

Thursday, July 17: 2 p.m., "Electrocardiography of the Arrhythmias II. Conduction Defects", Dr. E. Halliday; 3.30 p.m., clinic, Dr. J. Halliday and Dr. J. Palmer.

Friday, July 18: 1.15 p.m., post-graduate afternoon; 3.30 p.m. to 4.15 p.m., "Electrocardiographic Practice I", Dr. A. Seldon.

Monday, July 21: 2 p.m., "Electrocardiography of Coronary Disease I", Dr. A. Seldon; 3.30 p.m., "Pitfalls in Electrocardiographic Technique", Mr. W. Bell and Miss White; "Electrocardiographic Practice II", Dr. E. Halliday.

Tuesday, July 22: 2 p.m., "Electrocardiography of Coronary Disease II", Dr. E. Halliday; 3.30 p.m., clinic, Dr. K. Maddox, Dr. B. Sinclair-Smith.

Wednesday, July 23: 2 p.m., "Electrocardiography of Extra-cardiac Disease. Effect of Drugs, Electrolytes on Electrocardiogram *et cetera*. Pericarditis. *Cor Pulmonale*", Dr. B. Sinclair-Smith. 3 p.m., ward rounds, Dr. K. Maddox.

Thursday, July 24: 2 p.m., "Electrocardiography of Childhood and Congenital Heart Disease", Dr. R. Blacket. 3.30 p.m., clinic, Dr. J. H. Halliday, Dr. J. Palmer.

Friday, July 25: 2 p.m., post-graduate afternoon. 3.30 p.m. to 4.15 p.m., "Electrocardiographic Practice III", Dr. A. Seldon.

Those wishing to attend this course who are not members of the staff of the Royal Prince Alfred Hospital must register with the Course Secretary, Post-Graduate Committee in Medicine, 131 Macquarie Street, Sydney. Fee: £6 6s.

Congresses.

CONFERENCE OF THE NEW ZEALAND BRANCH OF THE BRITISH MEDICAL ASSOCIATION.

THE biennial conference of the New Zealand Branch of the British Medical Association is to be held in Auckland, New Zealand, from February 10 to 13, 1953. Members of the British Medical Association in Australia are welcome to attend.

DISEASES NOTIFIED IN EACH STATE AND TERRITORY OF AUSTRALIA FOR THE WEEK ENDED JUNE 7, 1952.[1]

Disease.	New South Wales.	Victoria.	Queensland.	South Australia.	Western Australia.	Tasmania.	Northern Territory.	Australian Capital Territory.	Australia.
Acute Rheumatism	..	..	..	..	..	..	..	..	..
Amœbiasis	..	..	..	..	1(1)	..	..	..	1
Ancylostomiasis	..	..	..	..	1(1)	..	..	..	1
Anthrax	..	..	..	..	..	..	..	..	..
Bilharziasis	..	..	..	..	..	..	..	..	..
Brucellosis	..	..	..	..	..	..	..	..	..
Cholera	..	..	..	..	..	..	..	..	..
Chorea (St. Vitus)	..	..	..	..	..	..	..	..	..
Dengue	..	..	..	..	..	..	..	..	..
Diarrhœa (Infantile)	..	..	6(5)	..	..	..	..	..	6
Diphtheria	22(5)	8(8)	4(2)	1	5(5)	..	..	..	40
Dysentery (Bacillary)	..	..	3(3)	..	..	..	..	..	3
Encephalitis	..	3(1)	..	..	..	..	..	..	3
Filariasis	..	..	..	..	..	..	..	..	..
Homologous Serum Jaundice	..	..	..	..	..	..	..	..	..
Hydatid	..	2(1)	..	..	..	..	..	..	2
Infective Hepatitis	..	..	..	..	6(4)	..	..	..	6
Lead Poisoning	..	..	..	..	..	..	..	..	..
Leprosy	..	..	..	..	..	..	1	..	1
Leptospirosis	..	..	6(1)	..	..	..	..	..	6
Malaria	..	..	..	..	..	..	..	..	..
Meningococcal Infection	2(2)	3(2)	..	2	..	1	..	..	8
Ophthalmia	..	..	..	..	..	..	..	..	..
Ornithosis	..	..	..	..	..	..	..	..	..
Paratyphoid	..	..	..	..	..	..	..	..	..
Plague	..	..	..	..	..	..	..	..	..
Poliomyelitis	13(6)	6(4)	..	9(4)	1	..	..	..	29
Puerperal Fever	..	..	..	..	..	..	..	..	..
Rubella	..	4(4)	..	..	..	..	..	..	4
Salmonella Infection	..	..	..	..	..	..	..	3	3
Scarlet Fever	21(9)	24(13)	7(5)	3(3)	3(3)	9(2)	..	..	67
Smallpox	..	..	..	..	..	..	..	..	..
Tetanus	..	1(1)	2(1)	..	..	..	..	..	3
Trachoma	..	..	..	..	..	..	..	..	..
Trichinosis	..	..	..	..	..	..	..	..	..
Tuberculosis	32(22)	23(21)	14(6)	7(5)	11(6)	2	1	1	91
Typhoid Fever	..	..	..	1	..	..	..	..	1
Typhus (Flea-, Mite- and Tick-borne)	..	..	2	..	1(1)	..	..	..	3
Typhus (Louse-borne)	..	..	..	..	..	..	..	..	..
Yellow Fever	..	..	..	..	..	..	..	..	..

[1] Figures in parentheses are those for the metropolitan area.

AN HONOUR FOR SIR VICTOR HURLEY.

SIR VICTOR HURLEY, K.B.E., C.B., C.M.G., M.D., F.R.C.S., F.R.A.C.S., of Melbourne, has been elected an Honorary Fellow of the Association of Surgeons of Great Britain and Ireland. We congratulate Sir Victor Hurley on this honour, which has been extended to only a small group of eminent Australian surgeons.

Obituary.

ERNEST WINBOLT LEWIS.

WE regret to announce the death of Dr. Ernest Winbolt Lewis, which occurred on June 21, 1952, at Box Hill, Victoria.

GILBERT ELLIOT AITKEN.

WE regret to announce the death of Dr. Gilbert Elliot Aitken, which occurred on June 22, 1952, at Adelaide, South Australia.

Australian Medical Board Proceedings.

NEW SOUTH WALES.

THE following have been registered, pursuant to the provisions of the *Medical Practitioners Act*, 1938-1950, of New South Wales, as duly qualified medical practitioners:

Yorkston, Neil James, M.B., B.S., 1952 (Univ. Sydney).

Clapp, Gilbert Edward Spence, M.B., B.S., 1950 (Univ. Melbourne); Crotty, John Martin, M.B., B.S., 1950 (Univ. Melbourne); Elkington, Edward James, M.B., B.Ch., 1951 (Univ. Oxford); Giri, Devarayadoorg Venkata, L.R.C.P. (Edinburgh), L.R.C.S. (Edinburgh), L.R.F.P.S. (Glasgow), 1908, D.P.H. (London), 1910, D.O. (Oxford), 1912; Lawson, Wilfrid Ernest Little, M.B., Ch.B., 1928 (Univ. Leeds); O'Leary, Timothy Joseph, M.B., B.Ch., 1949 (Nat. Univ. Ireland); Rosten, Morris, M.R.C.S. (England), L.R.C.P. (London), 1941; Sacks, Marcus Lionel, M.B., B.S., 1946 (Univ. London); St. John, John Michael Spenser, M.R.C.S. (England), L.R.C.P. (London), 1948; Streeten, George Edward Wardell, M.B., B.S., 1945 (Univ. Queensland).

Easy, John Edmund, M.B., B.S., 1952 (Univ. Sydney); Edgley, Robert Steel, M.B., B.S., 1952 (Univ. Sydney); Edwards, Ian Swales, M.B., B.S., 1952 (Univ. Sydney); Edye, Beryl Vivienne, M.B., B.S., 1952 (Univ. Sydney); Ellard, John Henry Temple, M.B., B.S., 1952 (Univ. Sydney); Emmerson, Bruce Raymond McKevitt, M.B., B.S., 1952 (Univ. Sydney); Eskdale, Walter John, M.B., B.S., 1952 (Univ. Sydney); Evans, Patricia Isobel, M.B., B.S., 1952 (Univ. Sydney).

Farrell, John Campbell, M.B., B.S., 1952 (Univ. Sydney); Feather, Robert Hartley, M.B., B.S., 1952 (Univ. Sydney); Findlay, James Scott, M.B., 1952 (Univ. Sydney); Fisher, June Ida Wyndham, M.B., B.S., 1952 (Univ. Sydney); Fisher, Roderick William, M.B., B.S., 1952 (Univ. Sydney); Fisher, Wallace George, M.B., B.S., 1952 (Univ. Sydney); Fitzgerald, Kevin Joseph, M.B., B.S., 1952 (Univ. Sydney); Flynn, Michael John, M.B., B.S., 1952 (Univ. Sydney); Ford, Lorna Mary, M.B., B.S., 1952 (Univ. Sydney); Francis, Peter Newton, M.B., B.S., 1952 (Univ. Sydney); Fuller, Kenneth Thomas, M.B., B.S., 1952 (Univ. Sydney).

The following additional qualifications have been registered: Goldie, John Ernest Dunlop (M.B., B.S., 1945, Univ. Sydney), F.R.C.S. (England), 1951; McGrath, David Henry (M.B., 1942, Univ. Sydney), B.S., 1945 (Univ. Sydney), M.R.C.O.G., 1951; Rhydderch, Alun Iorwerth (M.B., B.S., 1941, Univ. Sydney), F.R.C.S. (England), 1951.

Medical Appointments.

Dr. J. Edis has been appointed government representative on the Committee of Management of the Peacock Convalescent Hospital, Tasmania.

Dr. Alan Davison, junior, has been appointed a quarantine officer under the provisions of the *Quarantine Act*, 1908-1950.

Dr. T. J. Flynn has been appointed government medical officer at Richmond, Queensland.

Dr. L. J. Jones and Dr. J. K. Wilson have been appointed deputy medical superintendents in the Division of Mental Hygiene, New South Wales.

Nominations and Elections.

THE undermentioned has applied for election as a member of the New South Wales Branch of the British Medical Association:

Lines, Edward John, M.B., B.S., 1952 (Univ. Sydney), Pennant Hills Road, Carlingford, New South Wales.

Diary for the Month.

JULY 8.—New South Wales Branch, B.M.A.: Executive and Finance Committee.

JULY 8.—New South Wales Branch, B.M.A.: Organization and Science Committee.

JULY 11.—Queensland Branch, B.M.A.: Council Meeting.

JULY 14.—Victorian Branch, B.M.A.: Finance Subcommittee Meeting.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Medical Secretary, 135 Macquarie Street, Sydney): All contract practice appointments in New South Wales.

Victorian Branch (Honorary Secretary, Medical Society Hall, East Melbourne): Associated Medical Services Limited; all Institutes or Medical Dispensaries; Australian Prudential Association, Proprietary, Limited; Federal Mutual Medical Benefit Society; Mutual National Provident Club; National Provident Association; Hospital or other appointments outside Victoria.

Queensland Branch (Honorary Secretary, B.M.A. House, 225 Wickham Terrace, Brisbane, B17): Brisbane Associated Friendly Societies' Medical Institute; Bundaberg Medical Institute. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

South Australian Branch (Honorary Secretary, 178 North Terrace, Adelaide): All Contract Practice appointments in South Australia.

Western Australian Branch (Honorary Secretary, 205 Saint George's Terrace, Perth): Norseman Hospital: all Contract Practice appointments in Western Australia. All government appointments with the exception of those of the Department of Public Health.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

All communications should be addressed to the Editor, THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, New South Wales. (Telephones: MW 2651-2.)

Members and subscribers are requested to notify the Manager, THE MEDICAL JOURNAL OF AUSTRALIA, Seamer Street, Glebe, New South Wales, without delay, of any irregularity in the delivery of this journal. The management cannot accept any responsibility or recognize any claim arising out of non-receipt of journals unless such notification is received within one month.

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